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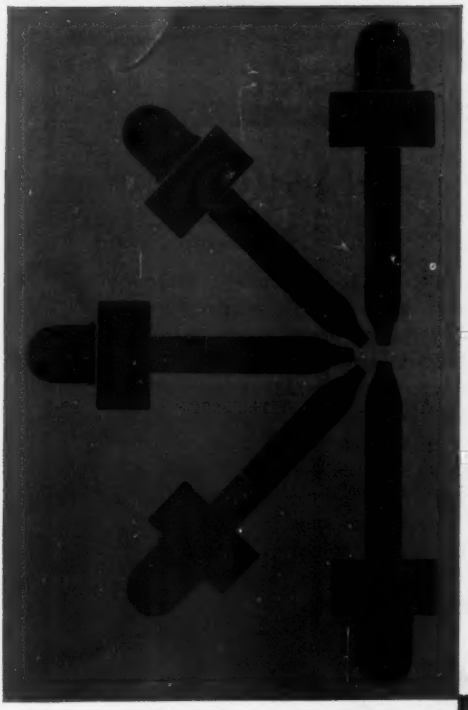

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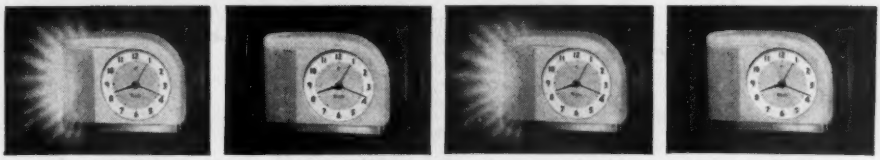

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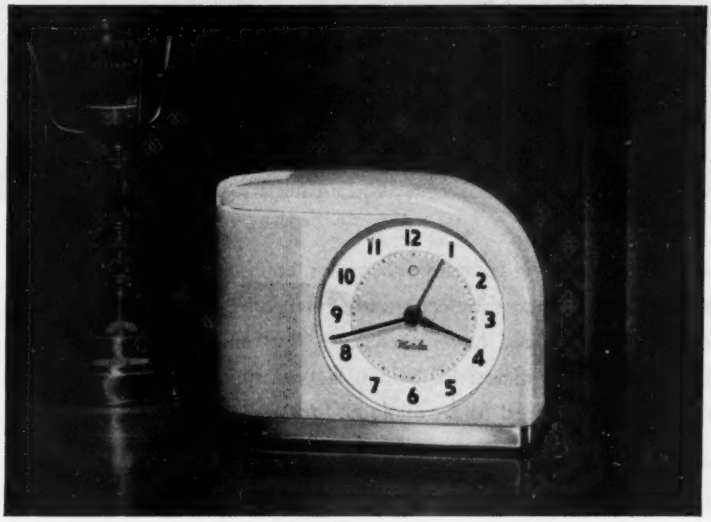

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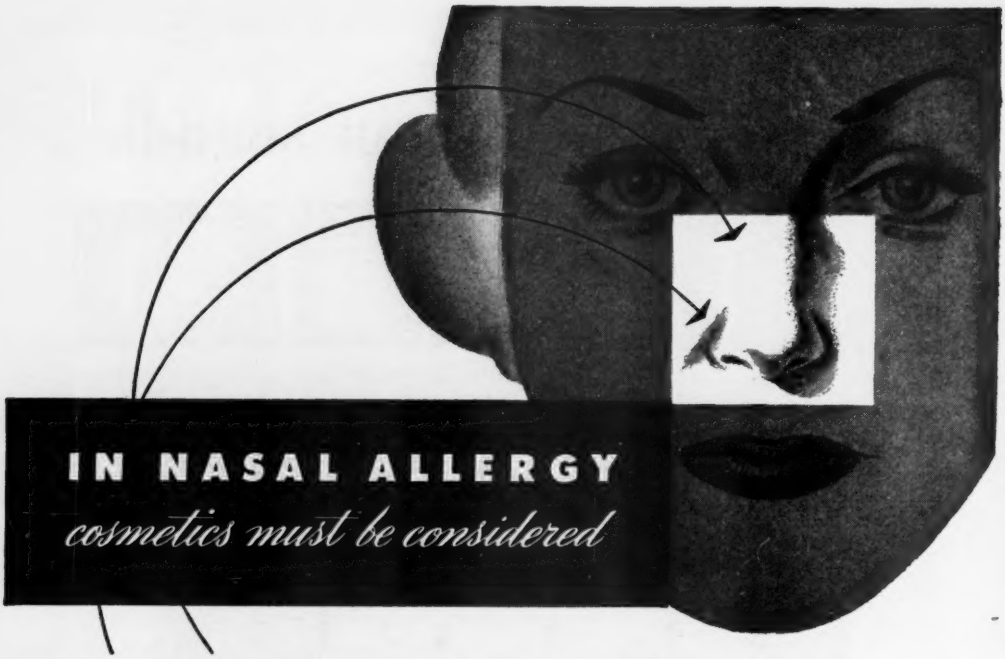

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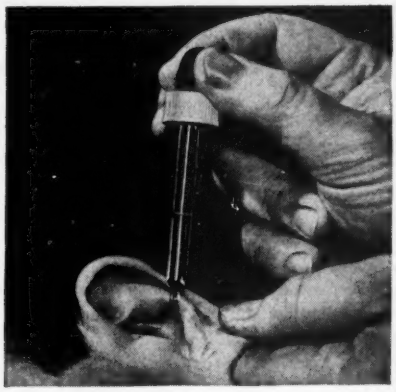

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*Laryngoscope 58:1279 (Dec.) 1948.

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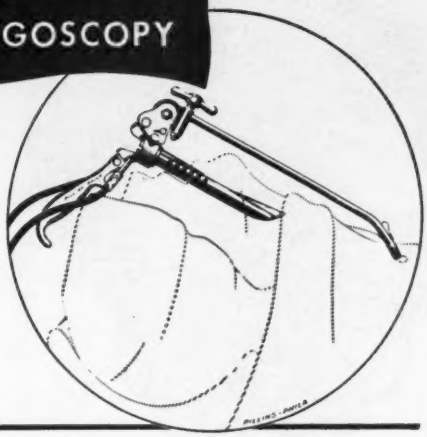

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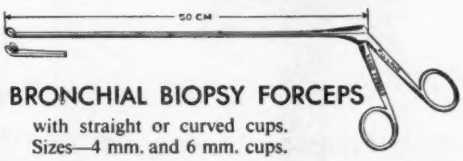

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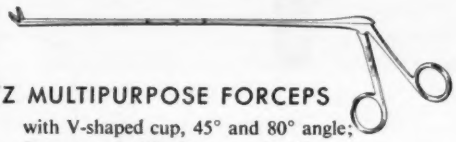

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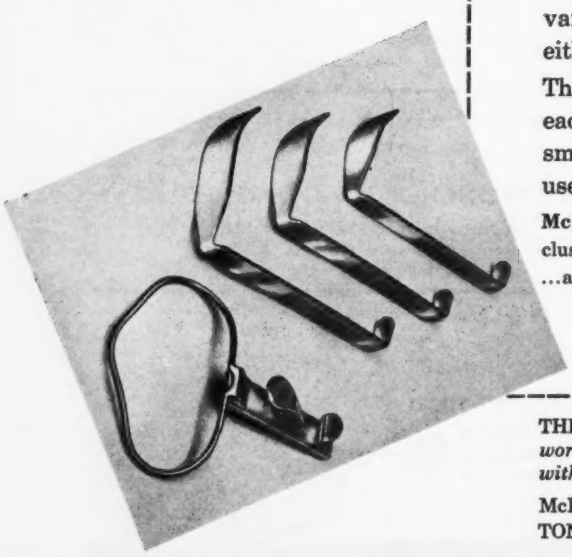

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ARCHIVES OF OTOLARYNGOLOGY

VOLUME 58 JULY 1953 NUMBER 1

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PRIMARY TRACHEAL TUMORS IN THE INFANT AND ADULT

JOSEPH G. GILBERT, M.D.
LAURENCE A. MAZZARELLA, M.D.
AND
LOUIS J. FEIT, M.D.
BROOKLYN

TUMORS having their origin in the trachea are rare. This article is a study of such tumors as they occur in the infant and the adult.

INCIDENCE

Our compilation (including our cases) revealed a total of 546 cases of primary tracheal tumors in both infants and adults (Table 1). Of this number, 509 (92.1%) occurred in adults. In a previous presentation, in 1949,[1] we dealt with the occurrence of such tumors in infants and children. We were able to find but 39 cases reported in the literature up to that time. To these we added 2 of our own, making a total of 41. Since then, 2 more have been reported, making a total of 43 tumors in infants and children (7.9%).

PATHOLOGY

A striking difference exists in the types of tumors that appear in children and in adults. In adults, of 503 tumors, 247 (49.1%) were malignant and only 256 (50.9%) nonmalignant. In children, 40 (93.1%) were nonmalignant, and only 3 (6.9%) were malignant.

It is significant that of 247 malignant tumors in adults, 194 (78.5%) were carcinomatous, whereas the 3 reported malignant tumors in infants were sarcomatous and all occurred in girls (Tables 2 and 3).

Of the 256 benign tumors in adults, the predominant types were some form of osteochondroma in 76 (29.4%), papilloma in 42 (16.4%), and fibroma in 30 (11.7%). In children the predominant benign tumors were papilloma in 23 (57.5%), fibroma in 9 (22.5%), and angioma in 6 (15%) (Table 4).

Because we have been unable to find any previous compilation of primary tracheal tumors occurring in children, a detailed study of that type appears in Table 5. This table includes cases reported up to August, 1952.

From the State University of New York College of Medicine at New York, Department of Otolaryngology, Service of Dr. J. G. Gilbert.

1. Gilbert, J. G.; Kaufman, B., and Mazzarella, L. A.: Tracheal Tumors in Infants and Children, J. Pediat. **35**:63-69, 1949.

TABLE 1.—*Primary Tracheal Tumors in Children and Adults*

Type of Tumor	Von Bruns, 1898 *	Krieg, 1908 *	Lombard and Baldenweck, 1914 †	D'Aunoy and Zoeller, 1931 ‡	Culp, 1938 §	Authors, Aug., 1952 ‖
Carcinoma	31	40	54	91	147	194
Chondroma, osteoma, tracheopathia osteoplastica	29	42	47	65	71	77
Papilloma	33	41	51	59	63	65
Fibroma	23	25	29	33	36	39
Sarcoma	14	21	23	26	31	39
Intratracheal goiter	7	14	19	25	28	32
Adenoma	5	6	6	8	9	14
Angioma	..	..	1	2	2	8 ¶
Mixed salivary tumor	..	..	..	2	3	7
Basal-cell carcinoma	..	..	..	..	..	6
Cylindroma	..	..	..	1	1	6 ‡
Fibroepithelioma	..	..	..	..	..	5
Lipoma	3	4	5	5	5	5
Amyloid tumor	..	..	1	2	2	4
Lymphoma	2	2	2	3	4	4
Osteogenic sarcoma	..	..	..	..	..	3
Leiomyoma	..	..	..	..	..	2
Endothelioma	..	..	..	1	1	2 **
Adenocarcinoma	..	..	..	..	..	2
Carcinosarcoma	..	..	..	1	1	1
Rhabdomyoma	..	..	..	..	..	1
Oncocytoma	..	..	..	..	..	1
Type histologically undetermined	..	6	14	27	29	29

* For reference, see Table 5. Cases include those collected up to that year.
† Lombard, P., and Baldenweck, A. L.: Ann. mal. de l'oreille, du larynx **40:** 491, 1914.
‡ D'Aunoy, R., and Zoeller, A.: Arch. Path. **11:** 589-600. Cases include those collected up to that year.
§ Culp, O. S.: J. Thoracic Surg. **7:** 471-487, 1938. Cases include those collected up to that year.
‖ Cases collected up to that year.
¶ Includes authors' two cases.
‡ Includes authors' case.
** Includes authors' case.

TABLE 2.—*Incidence of Primary Tracheal Tumors in Children and Adults*

	Children	Adults
Nonmalignant	40 (93.1%)	256 (50.9%)
Malignant	3 (6.9%)	247 (49.1%)
Total	43	503

TABLE 3.—*Predominant Malignant Primary Tracheal Tumors in Children and Adults*

	Children	Adults
Sarcoma	3 (100%)	36 (14.5%)
Carcinoma	0	194 (78.5%)

TABLE 4.—*Predominant Benign Primary Tracheal Tumors in Children and Adults*

	Children	Adults
Osteochondroma	1 (2.5%)	76 (29.4%)
Papilloma	23 (57.5%)	42 (16.4%)
Fibroma	9 (22.5%)	30 (11.7%)
Angioma	6 (15.0%)	2 (0.78%)

SITE AND SYMPTOMATOLOGY

The most frequent site of the tumor in the adult has been the lower third of the trachea, followed by the upper third, and least common is the middle third. From the meager information available it appears that the most frequent site in infants and children is the upper third, the inferior third being a poor second.

Symptomatology depends on (1) site of the tumor, (2) type of attachment, and (3) size of the tumor and width of the trachea.

1. *Site of the Tumor.*—Tumors arising in the lower third of the trachea and growing down into either bronchus give rise to signs of partial or complete atelectasis in addition to wheezing or harsh breath sounds. If the tumor is located in the upper third, interfering with the action of the vocal cords, noisy respirations and difficulty in breathing ensue. Tumors in the middle third are less likely to cause severe symptoms and may go unrecognized.

2. *Type of Attachment.*—If the tumor is attached by a pedicle, as in one of our cases, it may ascend on expiration and descend on inspiration. Pedunculated tumors in the lower third may plug a bronchus during inspiration and give rise to atelectasis, whereas on expiration they cause fewer symptoms. A similar type of attachment in the upper third of the trachea may cause few symptoms during inspiration but more on expiration. If the attachment has a broad base the symptoms are generally milder.

3. *Size of Tumor and Width of Trachea.*—Obviously, the larger the tumor the more the airway is compromised and the more prominent the symptoms. In the wider trachea of adults there can be a fair-sized tumor with few symptoms. (A 76-year-old woman with a tracheal endothelioma measuring 1 by 0.5 cm., attached by a small pedicle to the posterior wall of the lower third of the trachea, had no signs or symptoms referable to the trachea. The tumor was discovered while we performed a diagnostic bronchoscopy for pulmonary pathology.)

REPORT OF CASES

CASE 1.—S. A., a 23-year-old woman with a large cylindroma of the trachea, dated the onset of symptoms to the age of 17. At that time she experienced noisy respiration while sitting in a quiet place. Over a period of six years, the noise of respiration increased slightly and was particularly noticeable in quiet surroundings, and with it there was a gradual onset of dyspnea and hacking cough. The dyspnea became more evident at the age of 22, during her fifth month of pregnancy. Her obstetrician recognized her tracheal difficulty at that time, but the patient would do nothing about it. She went to term and was delivered of a healthy child. Subsequently she noticed that her household duties, including the care of her infant, were causing her to be excessively fatigued and dyspneic, making it necessary for her to rest at frequent intervals. Some eight months after her delivery, after an acute upper respiratory infection of three days' duration, she was brought into the emergency room of the Beth-El Hospital with severe dyspnea and cyanosis. An emergency tracheotomy was performed and it relieved her of cyanosis and dyspnea. On the following morning indirect laryngoscopy disclosed that the right cord was slightly elevated above the level of the left. On forced respiration and phonation, with the tracheotomy-tube lumen covered, the left cord moved well, but there was practically no motion of the right cord. Below the latter there could be seen an elevated mass occupying the right half of the tracheal wall. Engorged vessels were traversing its smooth surface. Three days later a biopsy specimen was taken from this mass by indirect laryngoscopy. (The tumor was first reported to be a mixed tumor of the salivary type and then, by another competent pathologist, a cylindroma.) Four days later when the patient's temperature was normal and her condition satisfactory, with intratracheal anesthesia induced by administration of the anesthetic through

TABLE 5.—*Primary Tracheal Tumors in Children*

Author	No. of Cases	Age	Sex	Type of Tumor	Site in Trachea	Symptoms	Treatment; Comment
von Bruns, P.: Handbuch für Laryngologie, Vienna, Alfred Holder, 1908, Vol. I.....	1	5	..	Fibrous polyp	.....	.....	.....
	1	12	M	Fibrous polyp	Lower third	.....	Death from blocked bronchus
	1	13	..	Fibrous polyp	.....	.....	.....
	16 *	1-10	..	Papilloma	Most on upper third of posterior wall	5 congenital cases, in hoarseness and dyspnea from birth	5 cases were congenital; 8 patients died of suffocation
Elsberg, C. A.: New York Med. Rec. **7**: 883, 1906..	1	10	F	Fibroma	Middle third, posterior wall	.....	.....
Krieg, E.: Beitr. klin. chir. **58**: 162-190, 1908.....	1	4	M	Papilloma	Multiple, all over trachea	.....	.....
	1	13	M	Papilloma	.....	.....	.....
	1	11	M	Papilloma	Middle third, anterior wall	.....	.....
	1	12	..	Chondro-osteoma	Multiple, in mucous membrane	.....	.....
MacKenzie, J., cited by Sauer.....	1	10	F	Fibroma	Upper third, anterior wall	.....	Operation and recovery
Sauer, W. E.: Laryngoscope **18**: 252-262, 1908....	1	1½	..	Fibroma	.....	.....	Death from suffocation
Beco, L.: Ann. mal. de l'oreille, du larynx, **34**: 450-451, 1908.....	1	10	M	Papilloma	.....	.....	Recurrent
	1	14	M	Papilloma	.....	.....	.....
	1	4	M	Papilloma	.....	.....	Multiple
Yankauer, S.: Ann. Otol. Rhin. & Laryng. **20**: 418, 1911.....	1	3	F	.....	Lower third, posterior wall	.....	No definite histologic diagnosis; tumor looked like cauliflower
Horgan, J. B.: Brit. M. J. **2**: 653, 1918.....	1	9	M	Fibroma	Above bifurcation; pedunculated	Dyspnea	Tracheotomy and excision
Dundas-Grant, J., and Perkins, J. J.: Proc. Roy. Soc. Med. (Sect. Laryng.) **16**: 7, 1922-1923.....	1	14	M	Papilloma	Entire trachea	Inspiratory dyspnea	Repeated operations; site healed

Watson-Williams, E.: Proc. Roy. Soc. Med. (Sect. Laryng.) **24:** 1637-1638, 1931.....	1	6	F	Sarcoma	Upper third	Dyspnea	Endoscopy and excision
Richards, L. G., and Dietrich, H. F.: Ann Otol. Rhin. & Laryng. **43:** 892-905, 1934.....	1	8 mo.	F	Fibrosarcoma	Upper third, anterior wall; 3.5 × 2 cm.	Wheezing, dyspnea, hoarse cries	Endoscopy and tracheotomy; child died; autopsy showed tumor below larynx
Abbatte, L.: Arch. Chronheilkunde **140:** 179, 1936	1	10	F	Sarcoma	Above bifurcation; posterior wall	Slowly increasing dyspnea	Removal with forceps and curet; radium treatment; no relapse after 9 mo.
Baldenweck, L., and Pouquet: Bronchoscop., oesophagoscop. et gastroscop. pp. 213-218, 1937	1	8 mo.	F	Fibroma	.....	.....	.....
Hofmann, L.: Ztschr. Hals.-nasen-u.Ohrenh. **44:** 435-440, 1938.....	1	2 mo.	M	Hemangioma	Upper third, posterior wall	Congenital stridor, increasing dyspnea	Diagnostic excision with severe hemorrhage; tracheotomy; operation; tracheal fissure; death from pneumonia and pleurisy
	1	2 mo.	F	Hemangioma	Upper third, left posterior wall	Congenital stridor, cyanosis	X-ray therapy; tracheotomy 3 wk. later; death from pneumonia
Crafoord, C., and Lindgren, A. G. H.: Acta chir. scandinav. **92:** 481-506, 1945.....	1	13	F	Fibroepithelioma	Above bifurcation; lower third	Increasing stridor for 2 yr.	Bronchoscopy; excision
Gilbert, Kaufman, and Mazzarella [1].....	1	14 mo.	M	Hemangioma	Lower third	Difficult respiration, fever, cyanosis	Laryngoscopy; bronchoscopic removal; cure
	1	11 mo.	F	Fibroangioma	Upper third; 0.3 × 0.2 × 0.1 cm., 0.6 × 0.3 × 0.2 cm.	Difficult respiration, stridor, hoarse brassy cry	Laryngoscopy; bronchoscopy; removal; cure
Sharp, H. S.: J. Laryng. & Otol. **63:**413-414, 1949	1	5 mo.	F	Hemangioma	Upper third, right posterior wall	Stridor from age of 3 wk.	Thyroid split, cricoid and 1st 4 rings of trachea removed; cure
Rutherford, I. M., and Sutherland, A. W.: New Zealand M. J. **50:** 570-571, 1951.....	1	4 mo.	..	Angioma	Upper third, left lateral wall	Stridor started at age of 8 wk.	Thyroid split, cricoid and 1st few rings of trachea removed; diathermy; cure at age of 6 mo.

[1] Cases collected to that date.

the tracheotomy tube, a laryngofissure was performed. In addition, the cricoid cartilage and the first two tracheal rings were cut in the midline. This maneuver was sufficient for exposure and removal of the tumor. It occupied the right half of the trachea, starting just below the cord and extending down almost to the third tracheal ring. The attachment, however, was of the button variety, with its main point of attachment on the lateral wall in the region of the cricoid and first tracheal ring. A No. 15 blade was used to separate the tumor from its attachment to underlying cartilage on the one hand and from the mucous membrane of the intercartilaginous areas on the other. This procedure was followed by electrodesiccation of the margins of the raw mucosa. A nasal feeding tube was used for three days postoperatively. Recovery was uneventful. Follow-up some 12 months after surgery disclosed a normal-functioning larynx with a good voice and no difficulty in swallowing. There appeared to be no evidence of regrowth of the tumor.

There are four salient points with reference to this case: 1. The gradual and almost imperceptible onset of symptoms, starting with noisy breathing and ending with severe dyspnea. 2. The type of tumor encountered. In spite of the fact that histologic studies of the biopsy and surgical specimens revealed the same characteristics, several competent pathologists reported the tumor to be either a mixed salivary tumor or a cylindroma. There is good reason for confusion in the histologic diagnosis of such tumors, since the histologic differences are only slight.[2] It does not fall within the scope of this paper to discuss the histologic differences. It is generally believed, however, that these tumors arise from the mucous glands of the trachea (or from the bronchus in the bronchial types). Reference to Table 1 discloses that seven mixed salivary tumors of the trachea and six cylindromas (including that in our case) have been reported in the literature. The tumor in our case, therefore, falls into a very rare group of primary tumors found in the trachea. 3. The operative approach used. A laryngofissure, including splitting of the cricoid and the first few rings of the trachea, was necessary for total removal of the mass. In spite of this approach, there remained a normal-functioning larynx, airway, and swallowing mechanism. 4. Recurrence of mixed salivary tumors. These tumors, according to Ward and Hendrick,[3] if incompletely removed recur immediately or after a period of many months or years, the frequency of recurrence being as high as 35 to 40%. It is therefore important in removing such tumors of the trachea that the mass, including the mucous membrane lining the inner surface of the cartilages as well as the intercartilaginous areas, be completely removed, as well as several millimeters of mucosa beyond the limits of the tumor itself. Electrodesiccation of the margins of the raw surface is also advisable. Care should be taken to avoid perforating the trachea. However, such perforation can be repaired with a graft.

CASE 2.—E. S., a boy aged 11 months, was admitted to Kings County Hospital on Oct. 29, 1948, with a two-day history of fever and difficulty in breathing. A diagnosis of mild laryngotracheobronchitis was made, and penicillin and sulfadiazine therapy was instituted. The child's temperature fell from 103 to 99 F., but he became "croupy" and cyanotic. On Nov. 1 he was transferred to Kingston Avenue Contagious Hospital. He breathed with difficulty, with marked suprasternal, infrasternal, and epigastric retractions, and was very restless.

On digital examination of the pharynx and hypopharynx no obstruction was palpated. Inspection of the throat revealed moderately enlarged tonsils. Laryngoscopy followed by

2. Belsey, R. H. R., and Valentine, J. C.: Cylindromatous Mucous-Gland Tumors of the Trachea and Bronchi: A Report of 3 Cases, J. Path. & Bact. **63**:377-387, 1951.

3. Ward, G. E., and Hendrick, J. W.: Diagnosis and Treatment of Tumors of the Head and Neck, Baltimore, Williams & Wilkins Company, 1950.

bronchoscopy revealed a markedly injected larynx, the cords moving freely. The tracheal mucosa was found to be irregularly raised and injected and in places covered with tenacious mucus. (The tumor was not recognized at that time because of the inflammatory reaction and mucus covering the tracheal wall.) The bronchi were clear. No foreign bodies were encountered.

Since the child grew progressively worse, tracheotomy was performed on the day of admission. The child gradually improved. On Nov. 10 decannulation was attempted, and because of a return of obstructive breathing recannulation was necessary. Decannulation was successfully accomplished on Nov. 14.

On Nov. 30, because of persistent slight stridor and noisy respiration, a 3.5-mm. bronchoscope was passed through a laryngoscope. The larynx was normal. At a point almost 3 cm. below the level of the cords (below the lowest end of the tracheal incision), a pedunculated, edematous mass attached to the anterior wall, moving downward on inspiration and upward on expiration, was seen. It occupied a little more than one-third of the lumen of the trachea at that site. A small foreign-body forceps being used, the tumor was removed with some difficulty. It was firm, with a smooth mucosa, and measured 0.3 by 0.2 by 0.2 cm. At the time of its removal it was considered to be a fibroma, and it was felt that the tumor's presence explained the noisy respiration present almost from the time of birth.

The specimen was a small oval piece of tissue removed from the trachea. Microscopic examination showed it to be composed of a loose stroma of fibrous connective tissue which supported numerous small channels lined by a single layer of well-preserved endothelial cells. No muscular wall could be distinguished, but in many places there was a well-defined, broad band of pink-staining hyaline material. Some of vascular spaces contained blood. Occasional small lymphocytes were seen within the stroma.

The diagnosis was capillary hemangioma of the tracheal wall.

The child's condition improved, and he was discharged on Dec. 12 in good condition. Examination on Jan. 21, 1949, showed him to be in excellent health; he had completely lost the noisy respiration present since birth.

DIFFERENTIAL DIAGNOSIS

Tracheal tumors in infants and children giving rise to repeated bouts of brassy cough, dyspnea, and inspiratory and expiratory difficulties, accompanied with suprasternal and infrasternal retractions, must be differentiated from laryngismus stridulus, cysts, and papillomas of the larynx.

In adults, asthma and chronic tracheitis and bronchitis can be mistaken for tracheal tumor.

In establishing a diagnosis of tracheal tumor, lateral roentgen studies of the neck may be of value in some instances. An angioma of the trachea was discovered in an infant by use of that technique (Figure). The best method, however, for discovering these tumors is direct laryngoscopy followed by tracheoscopy and biopsy studies.

TREATMENT

Many benign tumors can be removed through the bronchoscope. This procedure was carried out in our two cases in infants with hemangiomas of the trachea and also in the case of an adult with a small endothelioma. With more extensive tumors, it may be necessary not only to split the trachea but also to do a laryngofissure and split the cricoid in order to obtain sufficient exposure. This was the method used in the case of an adult with a cylindroma whose history we have described. It may be necessary before carrying out this extensive procedure to do a preliminary tracheotomy, thus overcoming dyspnea as well as providing an easy route for administration of intratracheal anesthesia. It should be pointed out at

this time that in the case of adenomas, mixed salivary tumors, or cylindromas, even though they are benign, because of their tendency to local recurrence after removal it is best to excise the normal mucosa several millimeters beyond the limits of the tumor. It appears advisable to electrodesiccate the site from which the tumor has been removed.

Malignant tumors of the trachea amenable to surgery require open operation on the trachea, including extirpation of the mass with the underlying cartilaginous rings. After this procedure, some sort of tracheoplasty for repair of the deficiency has to be done. Fascia lata has been sewn into place when only small deficiencies have occurred. However, when large areas have had to be covered, some authors [4]

Primary tracheal tumor (Case 2).

have used this graft plus a tantalum gauze support in order to fix the graft in place. In other instances,[5] full-thickness grafts taken from the inner aspect of the arm and reinforced with stainless steel gauze for support have been used. More recently, excision of the mass with end-to-end primary anastomosis has been performed with success.[6]

Roentgen therapy might be of value.

4. Cahan, W. G.: Carcinoma of Intrathoracic Trachea: Excision and Repair by Tantalum Gauze-Fascia Lata Graft; Report of a Case, J. Thoracic Surg. **23**:513-527, 1952.

5. Evans, B. H.: Myxochondroma of the Trachea: A Case Report, J. Thoracic Surg. **22**:585-591, 1951.

6. Kay, E. B.: Tracheal Resection with Primary Anastomosis, Ann. Otol. Rhin. & Laryng. **60**:864-870, 1951.

PROGNOSIS

Removal of benign tumors, either through the bronchoscope or by open operation, gives good results. However, it must be borne in mind that, in the case of so-called mixed salivary tumors, local growth may recur, and therefore meticulous care must be taken to effect complete removal at the initial operation.

In the case of malignant tumors, the earlier the tumor is recognized the better will be the chance for its total eradication. With the newer techniques of repair of tracheal deficiencies, more thorough and extensive procedures can be performed, including radical neck dissection for metastases to lymph nodes.

SUMMARY

Reported primary tracheal tumors occurring in both infants and adults were compiled from the literature. Our own experience with such tumors has been considered. Particular emphasis has been placed on the incidence and types of tumors encountered in the two groups. Prior to our presentation in 1949,[1] no article concerning these tumors in childhood had appeared. In our present compilation, through August, 1952, we found a total of 503 tumors in adults and only 43 in infants and children. Of the latter group only 3 were malignant (6.9%) and 40 (93.1%) were nonmalignant. On the other hand, of the tumors in adults, 49.1% were malignant and only 50.9% were nonmalignant. The predominant benign tumors in infants and children were the papilloma 23 (57.5%), fibroma 9 (22.5%), and angioma 6 (15%). In the adult the predominant benign tumors were the osteochondroma and tracheopathia osteoplastica, 76 (29.4%), followed by papilloma 42 (16.4%), and fibroma 30 (11.7%). As for the malignant tumors, it is significant to note that only three were reported in children, all of them occurred in infant girls, and all were sarcomatous. On the other hand, in adults, of 247 malignant growths, 194 (78.5%) were carcinomas and 36 (14.5%) were sarcomas.

A detailed study of infants with primary tracheal tumors reported in the literature appears in Table 5.

The symtomatology is discussed. It is pointed out that symptoms depend on (1) site of the tumor, (2) type of attachment, and (3) size of the tumor and width of the trachea. Surgical procedures, including endoscopic removal and open operation with tracheoplasty, are discussed.

CONCLUSION

Primary tracheal tumors in infants and children are of rather infrequent occurrence. Only 43, including 2 in cases of our own, have been reported in the literature. Data on a total of 503 tumors in adults have been compiled. In children approximately 93% of the tumors were benign, whereas in the adult approximately 50% were malignant. In the infant, the predominant malignant tumor was the sarcoma; in fact, this was the only malignancy reported, and, strangely enough, it was present only in girls. In the adult, carcinoma (almost 80%) is the predominant form of malignancy. The predominant benign tumors of childhood in the order of their frequency are the papilloma, fibroma, and angioma; those of the adult are the osteochondroma, papilloma, and fibroma.

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MULTIPLE PAPILLOMAS OF TRACHEA WITH MALIGNANT DEGENERATION

Report of Two Cases

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ALTHOUGH papilloma of the larynx is not an uncommon finding, primary papilloma of the trachea is infrequent and papillomatosis of the trachea, particularly in adults, is rare. Gilbert, Kaufman, and Mazzarella,[1] in reviewing the literature up to 1949, compiled a total of 529 primary tumors of the trachea.

The first recorded observation of a tracheal tumor was that of Lieutaud, in 1767, who discovered at necropsy a benign tumor believed to be a fibroma.[2] Turck, in 1861, was the first to observe a tracheal growth in a living person by indirect laryngoscopy.[2] In 1897, Killian visualized and diagnosed a tracheal tumor by endoscopy for the first time.[3]

Many kinds of tracheal tumors, both inflammatory and neoplastic, have been described since Lieutaud's original observation. The true neoplasms comprise a great variety of benign as well as malignant new growths. Of the benign neoplasms, papilloma is the most common. In a review of the literature up to 1947, Ellman and Whittaker [3] collected 67 cases of such tumors.

The following cases are noteworthy because they constitute exceedingly rare examples of carcinomatous degeneration in multiple papillomas of the trachea in adults.

REPORT OF CASES

CASE 1.—A 62-year-old white man was transferred to the hospital June 1, 1949, from a tuberculosis sanatorium, where he had undergone a complete clinical investigation for tuberculosis. No evidence of active pulmonary disease was found, but at bronchoscopy a large mass of papillomatous tissue was removed from the trachea. Upon admission he complained of extreme shortness of breath, tickling sensation in the throat, cough productive of blood-tinged sputum, general weakness, and a 15-lb. (6.8-kg.) weight loss. He had been well until six months prior to admission, when he noted difficulty in breathing, particularly on inspiration. The dyspnea gradually increased to the point where he was unable to work. The patient was cyanotic and in acute respiratory distress on admission. Respirations were rapid, shallow, and noisy. Breath

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1. Gilbert, J. G.; Kaufman, B., and Mazzarella, L. A.: Tracheal Tumors in Infants and Children, J. Pediat. **35**:63-69 (July) 1949.

2. Culp, O. S.: Primary Carcinoma of the Trachea, J. Thoracic Surg. **7**:471-487 (June) 1938.

3. Ellman, P., and Whittaker, H.: Primary Carcinoma of the Trachea, Thorax **2**:153-162 (Sept.) 1947.

sounds were decreased over the right chest. The voice was unchanged. Laboratory studies gave results within normal limits. Roentgenograms of the chest and neck revealed an irregularity in the suprasternal portion of the trachea compatible with a neoplasm. At bronchoscopy, multiple papillomas were seen on the posterior and lateral walls of the trachea. After local excision of the tumors, the patient made a rapid recovery, and he was discharged on the 47th hospital day. He remained asymptomatic for several months, after which the dyspnea recurred and progressed until the patient was in a state of imminent asphyxia which necessitated emergency bronchoscopy.

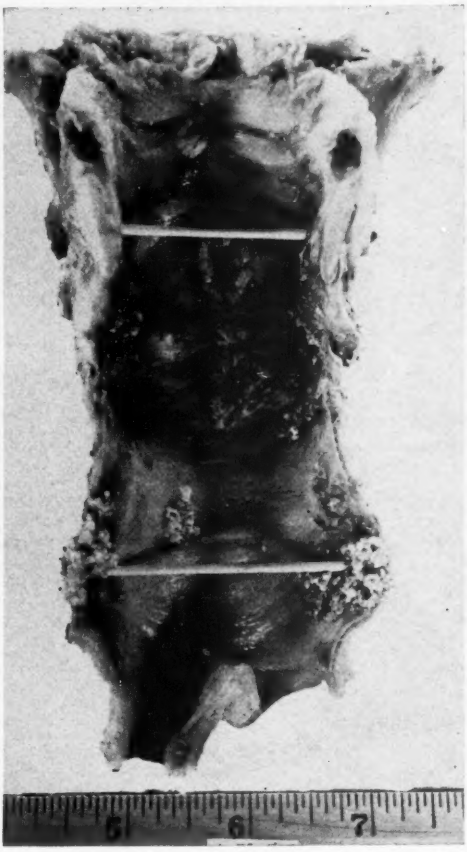

Fig. 1 (Case 1).—Larynx and trachea opened along posterior midline to expose hyperplastic laryngeal mucosa, cauterized papillary remnants in region of tracheotomy incision, and several intact papillomas.

Immediate relief followed removal of a voluminous quantity of papillomatous tissue from the trachea. Three endoscopic examinations performed during the next four weeks yielded more papillomatous tissue. At the last bronchoscopy, two small papillomas similar to those in the trachea were found in the left main bronchus.

In an attempt completely to eradicate the tumor tissue, the trachea was exposed through a collar-type cervical incision. The original plan to resect the involved portion of the trachea was abandoned because of the finding of metastatic tumor in the peritracheal lymph nodes. The trachea was incised, and a large mass of papillomatous tissue was removed. A specially made

splint was then inserted into the trachea. While the splint was being checked endoscopically, the patient suddenly died.

Postmortem Observations.—Grossly, the body showed a recent collar-type surgical incision in the lower neck and a longitudinal 4-cm. incision in the anterior wall of the trachea. Within the trachea, several widely separated, soft, gray, cauliflower-like tumors were seen. One, measuring 10 by 7 mm., was located on the left anterolateral wall 3.5 cm. above the carina, and a larger one was situated on the posterior wall 1 cm. above the carina. In a zone 3 to 5 cm. below the cricoid cartilage, the lumen was slightly constricted, and the mucosa in this area was roughened and discolored by cauterization. The tumor penetrated all layers of the wall in this region. In

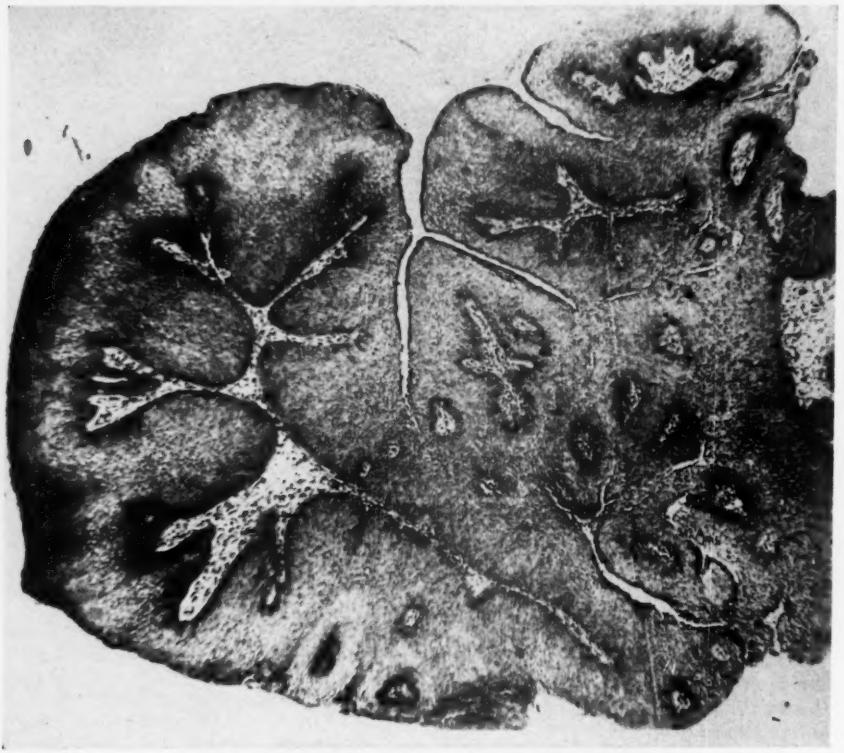

Fig. 2 (Case 1).—Photomicrograph (× 80) of part of papilloma showing characteristic papillary structure.

addition, two minute gray papillomas were found on the inferior margin of the right false cord near its lateral attachment. The bronchial mucous membranes were free of tumor (Fig. 1).

Microscopic examination revealed that all tumors consisted of a series of sessile papillae with thin vascular stalks covered by several to many layers of transitional or stratified squamous epithelium (Fig. 2). The cells were well differentiated and uniform except in one section, where they were enlarged, somewhat pleomorphic, and pale staining. The nuclei were large and contained giant nucleoli. Numerous mitoses, some of which were atypical, were also present (Fig. 3). The basement membrane was intact in all but a few sections, where there was invasion of the wall with separation or erosion of the tracheal rings (Fig. 4). The involved lymph node removed at operation showed extensive replacement of the lymphoid tissue by sheets and cords of well-differentiated squamous-cell carcinoma (Fig. 5).

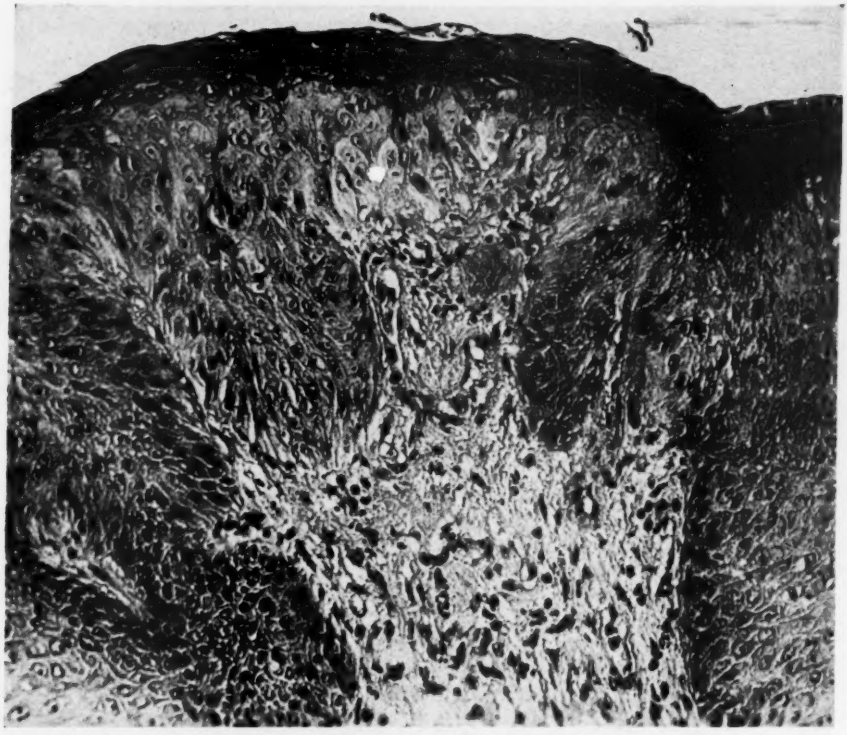

Fig. 3 (Case 1).—Photomicrograph (× 250) showing early malignant degeneration of papilloma.

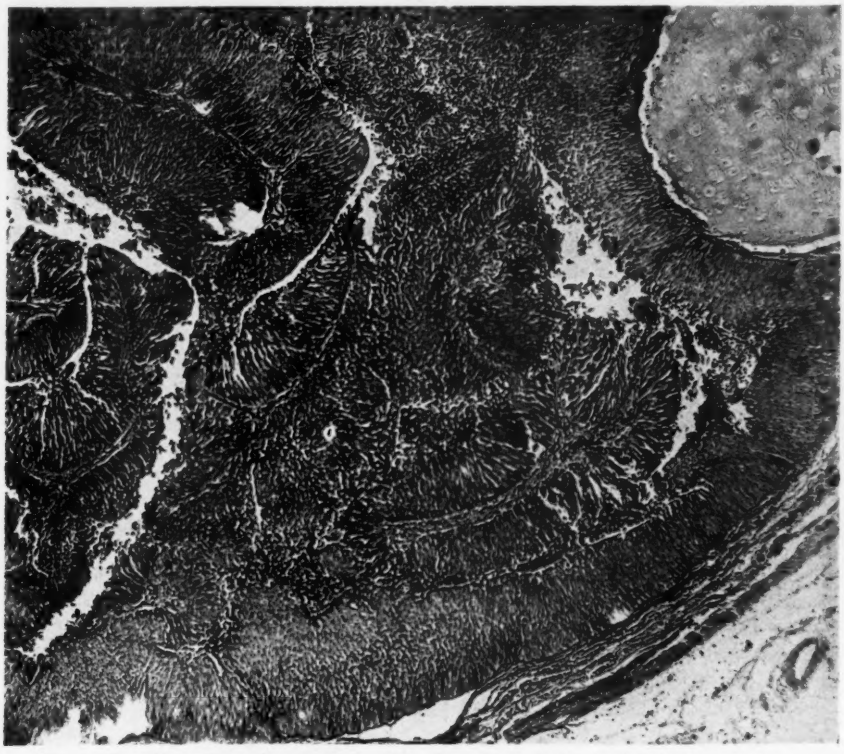

Fig. 4 (Case 1).—Photomicrograph (× 60) of trachea showing infiltration by papilloma.

A final diagnosis was made of papillomatosis of the trachea with carcinomatous degeneration and metastasis to the peritracheal lymph nodes.

CASE 2.—A 32-year-old white man was admitted to the hospital complaining of sharp pains in the lower left chest, chills, fever, and cough productive of blood-tinged sputum, all of two days' duration. He had had good health until six months before admission, when he acquired a persistent cough. One month prior to admission he received treatment in another hospital for pneumonia of the left lung. After discharge, he continued to cough and failed to regain his strength. Past history disclosed bilateral bronchopneumonia in 1942. An area of bronchopneumonia was demonstrated in the left lung by physical examination and confirmed by chest roentgenogram. Diplococcus pneumoniae was cultured from the sputum.

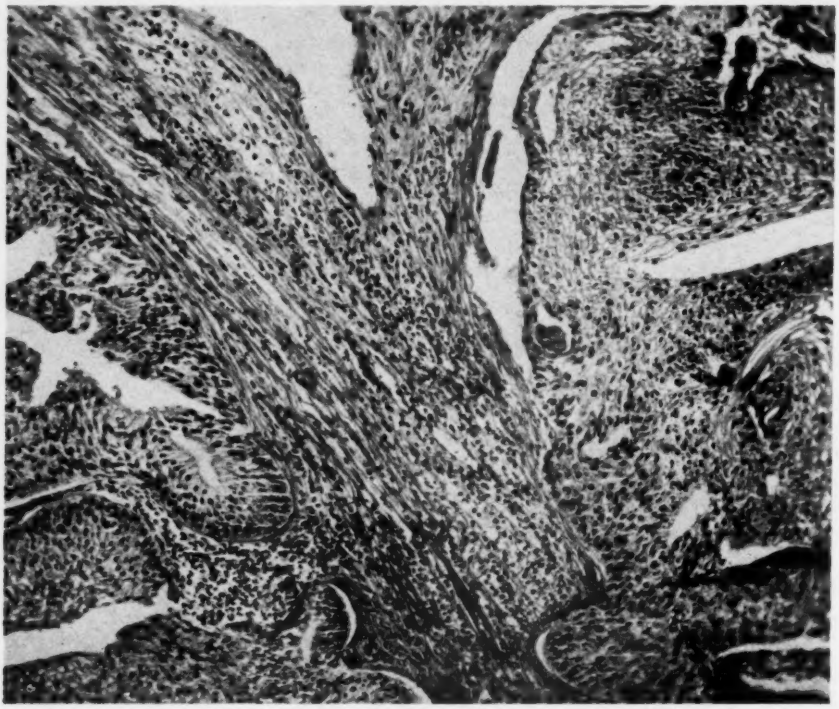

Fig. 5 (Case 1).—Photomicrograph (× 180) showing peritracheal lymph node invaded by well-differentiated neoplastic squamous and respiratory-tract epithelium.

Antibiotic therapy in one week produced prompt resolution of the pneumonia and disappearance of all signs and symptoms except hemoptysis. Because of persistent hemoptysis, a bronchoscopic examination was performed. The larynx was normal, but on the left posterior lateral wall of the trachea, 3 cm. above the carina, a soft, pink, papillary growth, 1 cm. in diameter, was found. The tumor, on biopsy, proved to be a typical papilloma (Fig. 6). The trachea was then explored through a left thoracotomy incision but failed to reveal infiltration of the wall or lymph node involvement. The remaining neoplastic tissue was, therefore, removed endoscopically. At later bronchoscopy, four small papillary-tissue fragments remaining at the site of the excision were removed. A diagnosis of low-grade squamous-cell carcinoma was made on microscopic examination by reason of the finding of small groups of atypical cells lying within the submucosa. Early malignant changes included pleomorphism, nuclear hyperchromatism, and dyskeratosis with early horn-pearl formation (Fig. 7).

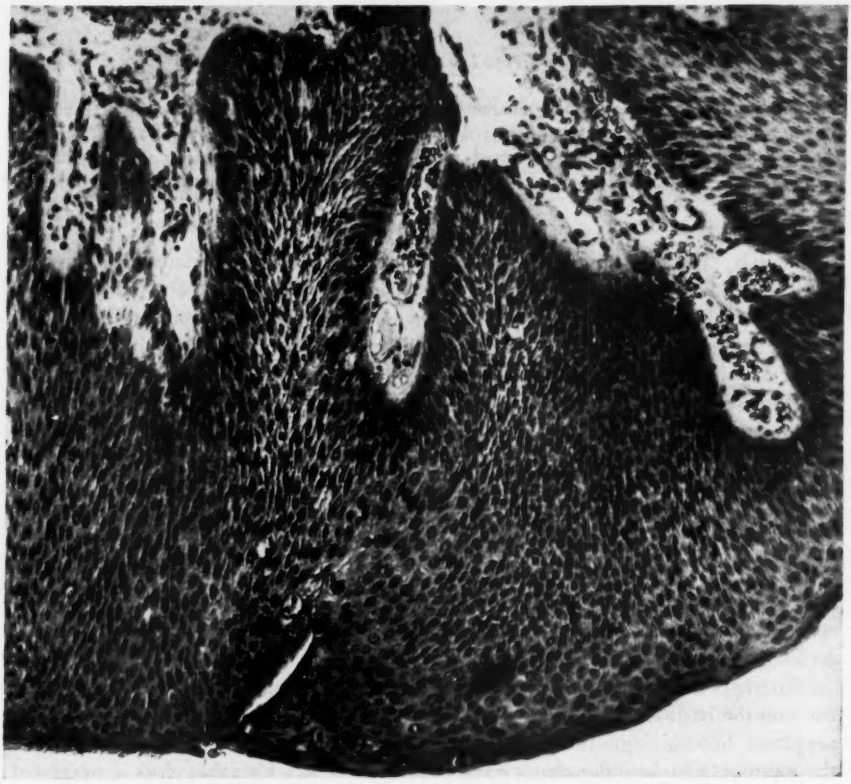

Fig. 6 (Case 2).—Photomicrograph (× 180) of papilloma showing characteristic stratified squamous epithelium.

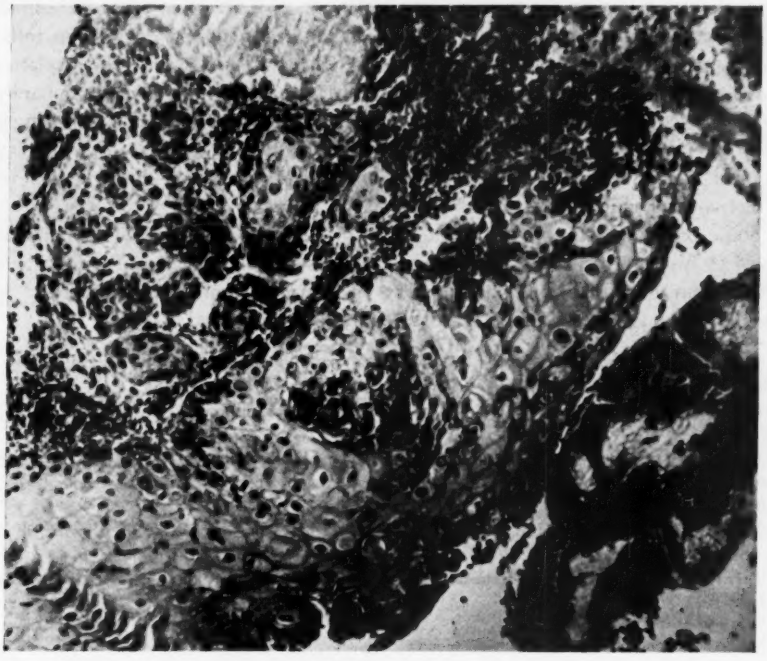

Fig. 7 (Case 1).—Photomicrograph (× 225) of part of papilloma showing dyskeratotic squamous epithelium accompanied by detached groups of dyskeratotic hyperchromatic epithelial cells in submucosa.

During a follow-up examination, another bronchoscopy was performed and a minute papillary growth was found on the left side of the trachea. Biopsy of the tumor disclosed a typical papilloma similar to those previously removed. After one week's hospitalization, the patient was discharged. He was asymptomatic and has remained in good health up to the time of writing.

COMMENT

Grossly, tracheal papillomas appear as sessile or low-pedunculated, cauliflower-like tumors with warty or mammillated surfaces. They are soft and often friable, bleeding easily with slight trauma. Their color varies from pale pink to red. They may be single but frequently are multiple, either grouped together to form a lobulated tumor or widely disseminated.

Microscopically, they show irregular, papillary, or villous processes composed of thin, branching stalks of loose, vascular fibrous tissue covered by several to many layers of well-differentiated epithelial cells. The cells are polyhedral to fusiform and arranged in a radially parallel or stratified fashion in the deeper layers, becoming more squamous at the surface. Cornification, when present, is seldom well developed. Nuclei are usually rounded but sometimes fusiform, moderately chromatic, and centrally located. A variable number of mitotic divisions are often present in the deeper layers. The basement membrane is regular and well defined. The subepithelial tissues, and even the epidermis, may be infiltrated with leucocytes if secondary inflammation is present.

Attempts have been made to explain the origin of papillomas of the respiratory tract on the basis of inflammation, congenital malformation, trauma, metaplasia, and neoplasia, but no single theory satisfactorily explains all cases. The possibility that these tumors may be inflammatory and probably due to a filterable virus is suggested by the work of Shope,[4] who demonstrated a papilloma-producing filterable virus in rabbits, which he was able to transmit readily to other rabbits. Ullmann [5] succeeded in transplanting laryngeal papilloma by inoculation of a filterable agent in both skin and mucous membranes of man and animal. He regards papilloma as an inflammatory reaction to a virus closely allied to that of skin warts. The relatively frequent reports in the literature of recurrence and dissemination after apparently adequate removal have been offered as evidence of an infectious nature. Blassingame and Jones [6] believe the papillomatosis in their case was the result of a neoplastic response to all respiratory epithelium, including the alveolar lining cells, to a papilloma-producing agent, probably a virus.

Some papillomas may have a congenital origin, as postulated by Kernan [7] with regard to his case of papilloma of the trachea associated with rupture of a tracheal diverticulum into the innominate artery in a 4-month-old infant. Priest [8] also considered the tracheal papilloma in his case to be congenital.

4. Shope, R. E., and Hurst, E. W.: Infectious Papillomatosis of Rabbits, with a Note on Histopathology, J. Exper. Med. **58**:607-624 (Nov.) 1933.

5. Ullmann, E. V.: On the Aetiology of the Laryngeal Papilloma, Acta oto-laryng. **5**:317, 1923.

6. Blassingame, C. D., and Jones, R.: Clinical Discussion (Papillomatosis of Larynx, Trachea, Bronchi and Pulmonary Alveoli), The John Gaston Hospital Clinical Pathological Conference, Memphis, July 10, 1950, J. Tennessee M. A. **43**:286-289 (Aug.) 1950.

7. Kernan, J. D.: Congenital Papilloma of the Trachea, Ann. Otol. Rhin. & Laryng. **45**:865-869 (Sept.) 1936.

8. Priest, R. E.: Tracheal Ectasia with Report of a Case, Ann. Otol. Rhin. & Laryng. **51**:1055-1061 (Dec.) 1942.

D'Aunoy and Zoeller [9] believe that trauma can be considered a possible predisposing etiologic factor, if not a direct cause, of papilloma. They offer as evidence for this hypothesis the rather frequent finding of papilloma around the tracheotomy orifice when a tracheotomy tube has been worn for a long period.

Implantation as an explanation for the occurrence of multiple papillomas has been accepted by most investigators. The frequent association of tracheal and laryngeal papillomas, their spontaneous disappearance and reappearance, and their strong tendency to recur at the same or different sites after apparently complete removal all suggest this concept. The precise method of spread, however, remains undiscovered. The most plausible explanation seems to be that of aspiration in which fragments of the primary tumor become detached and are carried to all parts of the respiratory tract. Hitz and Oesterlin [10] offer this explanation for the presence of nodules of stratified squamous epithelium in the lungs of a young child at necropsy. Buffmire, Clagett, and McDonald,[11] on the other hand, consider the papillomatosis of the larynx, trachea, and bronchi in their case to represent primary tumors of multicentric origin rather than aspirated neoplastic implants or embolic metastatic lesions.

Whether or not tracheal papillomas are initially produced by an inflammatory agent such as a virus, there seems little doubt about their eventual true neoplastic character. In both cases presented here growth occurred by infiltration as well as by surface spread, and in the first case metastases were found in the peritracheal group of lymph nodes. In the second case a follow-up over a number of years will be necessary to determine the ultimate outcome.

Jackson and Jackson [12] have stressed that even though the vast majority of papillomas are histologically benign, the term "benign" as applied to such tumors is not synonymous with "innocent." By virtue of their location and type of growth, these tumors may cause death, primarily by asphyxia due to respiratory obstruction or hemorrhage or secondarily by suppurative disease, such as pneumonia, abscess, bronchiectasis, or empyema, through obstruction to ventilation and drainage.

The paucity of physical findings of tracheal papilloma creates a difficult problem in early diagnosis. With uncomplicated tumors, signs and symptoms are absent until the growth reaches sufficient size to produce obstruction. The earliest sign, according to Jackson and Jackson,[13] is a wheeze heard at the open mouth, described as an asthmatoid wheeze. This eventually becomes a definite stridor and is associated with slowly increasing dyspnea. Later, tracheal obstruction results in secondary changes in the distal parts of the tracheobronchial tree, such as atelectasis, emphysema,

9. D'Aunoy, R., and Zoeller, A.: Primary Tumors of the Trachea: Report of a Case and Review of Literature, Arch. Path. **11**:589-600 (April) 1931.

10. Hitz, H. B., and Oesterlin, E.: A Case of Multiple Papillomata of the Larynx with Aerial Metastases to Lungs, Am. J. Path. **8**:333-338 (May) 1932.

11. Buffmire, D. K.; Clagett, O. T., and McDonald, J. R.: Papillomas of the Larynx, Trachea and Bronchi: Report of a Case, Proc. Staff Meet., Mayo Clin. **25**:595-600 (Oct. 11) 1950.

12. Jackson, C., and Jackson, C. L.: Benign Tumors and Tumor-like Conditions in the Tracheobronchial Tree, Am. J. Surg. **42**:275-281 (Oct.) 1938.

13. Jackson, C., and Jackson, C. L.: Benign Tumors of the Trachea and Bronchi, with Especial Reference to Tumor-like Formations of Inflammatory Origin, J. A. M. A. **99**:1747-1754 (Nov. 19) 1932.

pneumonia, bronchiectasis, and abscess. The usual course is one of increasing dyspnea terminating in asphyxia unless averted.

Papillomas cannot be distinguished with certainty from other tracheal tumors without bronchoscopic biopsy and histologic examination. The clinical features are diagnostic only of respiratory obstruction. The correct diagnosis can usually be made by following an orderly procedure which includes an accurate clinical history, a general physical examination, routine laboratory tests, roentgen-ray examinations, bronchoscopic examination, and biopsy with histologic study of the biopsy specimen.

Jackson [14] believes that all benign endotracheal tumors of limited extent require only bronchoscopic removal to be cured, with the exception of multiple papillomas. He advocates superficial excision of every tumor without further treatment of the base. Since these growths are prone to recur despite repeated excision and often do not recur at sites from which they were removed, it is useless and unnecessary and involves needless risk to attempt radical extirpation. It is apparent that for multiple papillomas, as illustrated in the cases reported here, more radical treatment is indicated. This observation is particularly true for adults who have large, extensive lesions that continue to recur after repeated removal. The type of therapy should be fitted to the individual case, but local excision in combination with fulguration or cauterization of the base with or without irradiation should be considered. Extratracheal exploration may occasionally be necessary to determine the presence or absence of lymph node metastasis. Thereafter, close observation over a prolonged period is necessary in order to detect recurrent or new papillomas and thereby prevent obstructive symptoms, pulmonary complications, and malignant degeneration.

SUMMARY

Two cases of multiple papillomas of the trachea in adults are reported. Evidence of malignant degeneration was found in each case, and in one metastasis to the peritracheal lymph nodes had occurred. The etiology, pathology, clinical features, diagnosis, and treatment are briefly discussed.

14. Jackson, C.: Tumors of the Trachea, South. Surgeon **5**:263-276 (Aug.) 1936.

CANCER OF THE LARYNX

Thirty-Year Survey at New Haven Hospital

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THERE were more than 260 cases in which cancer of the larynx was diagnosed at the New Haven Hospital during the 30-year period 1921-1951. In 235 of these the diagnosis was by either biopsy or postmortem examination, and these cases constitute the material discussed in this paper.

PREDISPOSING FACTORS

Chronological Distribution.—Whereas 94 cases were seen during the 20-year period between 1921 and 1940, 136 cases were seen during the 10-year period between 1941 and 1950.

Race, Religion, and Ethnic Groups.—The distribution of cases corresponds approximately to that found in the general population in this area; hence these factors do not appear to have any influence on the incidence of the disease.

Age and Sex.—The ages of patients in this series ranged from 21 to 90 years; 67% of the men were between 50 and 70, and 61% of the women were between 40 and 60 years of age.

Sex and Survival.—It is interesting to note that 50% of the women patients followed for at least five years survived five years or more but only 28% of the men followed for a similar period lived so long.

INCIDENCE

The factors predisposing to laryngeal cancer have been discussed by us in a previous publication [1] and are listed in Chart 1.

1. *Family History of Cancer.*—In the present series of 235 cases, 13% of the patients gave a history of cancer in one or more members of their families.

2. *Use of Alcohol.*—Patients were considered excessive drinkers who admitted to excessive intake or to more than three beers or two highballs or two shots of whiskey a day for an extended period of years. There were 69 patients, or 34%, who drank excessively (only one of these was a woman).

The Tumor Registry, New Haven Hospital, granted permission to use the follow-up data.

From Section on Otolaryngology, Department of Surgery, Yale University School of Medicine.

1. Malkin, J. S., and Kirchner, J. A.: Cancer of the Larynx in General Practice, Connecticut M. J. **16**:816-821 (Nov.) 1952.

3. *Use of Tobacco.*—The responsible carcinogenic agent is believed by some to be arsenic, which is present in minute amounts in the cigarette paper and is a constituent of the insecticides used on tobacco plants.[2] Patients whose chewing or smoking habits were labeled "excessive" or who smoked a pack or more of cigarettes a day for 10 years or more were included. One hundred one patients, or 43%, smoked or chewed to excess. Many more "moderate" smokers were not included.

4. *Industrial and Occupational Factors.*—In this series, metal working was the occupation in which the largest single group was engaged. Fifty-four, or 23%, of

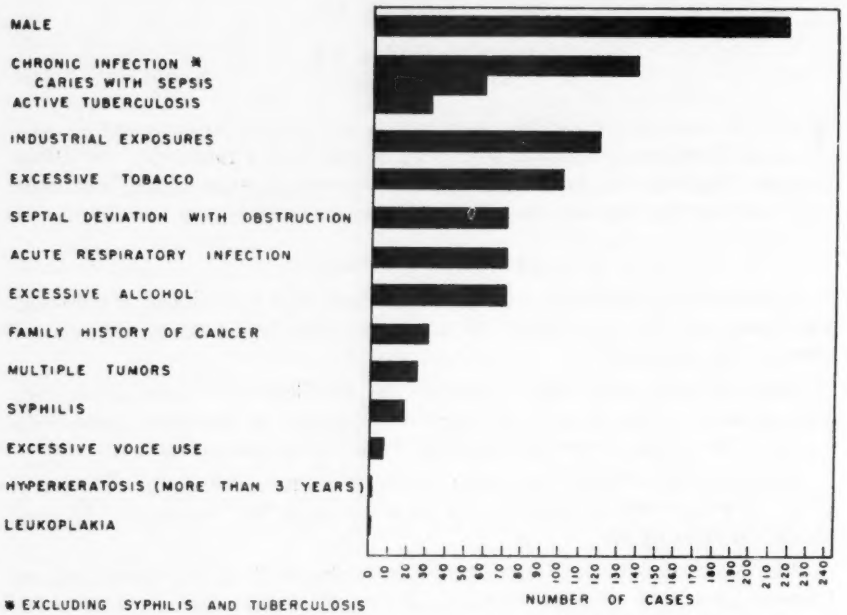

Chart 1.—Incidence of predisposing factors in 235 cases of cancer of the larynx.

the patients were metal workers of one type or another (tool and die makers, machinists, foundry workers, metal polishers, silver platers, blacksmiths, forge workers, smelters, etc.). The workers listed in Table 1 were exposed to iron, brass, silver fumes and dust, heat, petroleum oils, gasoline (benzine), ultraviolet and infrared rays (especially foundry workers), lead, zinc, pyridine and other proved irritants of the respiratory mucous membranes.[3] Occupations associated with oil, grease, tar, and benzine made up the next largest group. These totaled 7% of all occupations. Some of the remaining occupations listed in Table 1, each of which made up less than 4% of the total, are of interest. The exposures involved were varied and included rubber, arsenic (in insecticides), silica dust, lead, benzine,

2. Graham, E.: Some Questions About Bronchiogenic Carcinoma, New England J. Med. **245**:389-297 (Sept. 13) 1951.

3. Dublin, L. I., and Vane, R. J.: Occupational Hazards and Diagnostic Signs, Bulletin 582, U. S. Department of Labor, Bureau of Labor Statistics, 1942.

heat, organic dust, mercury, methyl alcohol, sulfuric acid, acetone, chromium, hydrofluoric acid, and others. Without exception, all these are considered marked irritants of the respiratory mucous membrane.[3]

5. *Predisposing Infection.*—Acute and chronic infections have been thought by several investigators to play a key role in the pathogenesis of laryngeal carcinoma.[4] The work of Winternitz on the pathology of influenza, in 1920, is of considerable interest in this respect.[4b] In studying the data listed in Table 2, we were surprised at the high incidence of tuberculosis. Thirty per cent of the patients had some form

TABLE 1.—*Significant Occupations in 235 Cases of Cancer of the Larynx*

Occupation	Cases		
	No.		%
Metal workers		54	23
Tool and dye makers	5		
Machinists	9		
Foundry workers	11*		
Moulders	4		
Metal polishers	7†		
Gunsmiths	3		
Other	15		
Occupations associated with oil, grease, and tar		16	7
Truckers	8		
Gas station attendants	2		
Mechanics	2		
Greasers	2		
Printers	1		
Roofers	1		
Factory workers (work unknown)		12	5
Farmers		9	4
Rubber workers		6	3
Hatters		5	2
Woodworkers		5	2
Stone workers		4‡	2
Housepainters §		2	1
Tobacco workers		2	1
Brewers		2	1
Cotton mill worker		1	1
Miscellaneous		83	..
Occupation unknown		34	..

* Nine were employed in a brass foundry; one worked in an iron foundry and had a concurrent pneumonoconiosis.

† One was a woman; one worked for 35 years polishing iron with an emery wheel; one polished silver for 26 years.

‡ Two were coal workers, one a cement worker, and one was a quarry worker. The quarrier, who had worked in a quarry 35 years, had pneumoconiosis.

§ One had a lead line.

of this disease. However, there was not a single one who had tuberculosis of the larynx. This applies also to syphilis, and it is quite understandable that these conditions were frequently erroneously diagnosed before cancer of the larynx became manifest. With respect to oral hygiene, the effect of a constant stream of infected saliva upon the larynx must be considered. It was found that markedly carious teeth were present in 100, or 47%, of the patients, and another 25% had oral sepsis in addition to the caries.

4. (*a*) Frissel, L. F., and Knox, L. C.: Primary Carcinoma of the Lung, Am. J. Cancer **30**:219-288 (June) 1937. (*b*) Winternitz, M. C., and others: The Pathology of Influenza, New Haven, Conn., Yale University Press, 1920.

6. *Nasal Obstruction.*—While tabulating our data, we were struck by the frequency with which septal deviation with obstruction was noted. Although we had not considered this as a possible carcinogenic factor, the work of Hilding [5] lends some weight to the idea that it may play a part. Hilding demonstrated that an increase in the flow of air in one nostril produced by closing the other surgically results in sqaumous metaplasia of the columnar epithelium in dogs and rabbits. This metaplasia is proportionate to the impact of the cold, dry air. If this condition existed for a long enough time, it is conceivable that metaplastic changes could occur in the mucous membrane of the larynx and lower respiratory tract. This problem is at present being investigated in our laboratory. In the present series of cases, 73 patients, or 31%, were noted to have septal deviation with obstruction,

TABLE 2.—*Acute and Chronic Infection Associated with Cancer of the Larynx*

Infection	Cases	
	No.	%
Acute		
Upper respiratory tract infection preceding onset	23	10
Pneumonia at onset	17	7
Past history of pneumonia	17	7
Influenza, 1918	13	6
Chronic		
Marked caries, alone	110	47
Marked caries, with oral sepsis	58	25
Tuberculosis, active and inactive	71	30
Syphilis	15	6
Chronic laryngitis (more than three years)	15	6
Chronic tonsillitis	12	5
Frequent severe upper respiratory tract infections	11	5
Sinusitis	9	4
Chronic rhinitis	7	3
Chronic cough (cause unknown)	7	3
Postnasal drip	6	3
Bronchiectasis	5	2
Chronic bronchial asthma	4	2
Chronic otitis	3	1
Frequent sore throats	1	1

and in 6 of these obstruction was bilateral. In many cases, unfortunately, the septum was not mentioned, and in others it was described only as "intact." In cases in which deviation was noted, the presence or absence of obstruction was often not recorded. These cases are not included in Chart 1.

7. *Excessive Use of Voice.*—In this series only 7 patients, or 3%, used their voices excessively (singing, preaching, etc.).

8. *Hyperkeratosis and Leucoplakia.*— These premalignant lesions were recorded in only one instance each before the discovery of laryngeal cancer. In many instances, however, hyperkeratosis was present histologically alongside epidermoid carcinoma at the time of diagnosis.

9. *Sex.*—The predominance of laryngeal cancer in the male is well demonstrated by the 12: 1 ratio found in this series; 217 of the patients were male, and 18, female.

5. Hilding, A.: Experimental Surgery of the Nose and Sinuses: I. Changes in the Morphology of the Epithelium Following Variations in Ventilation, Arch. Otolaryng. **16**:9-18 (July) 1932.

SYMPTOMS

Patient usually complained of more than one symptom. These symptoms are listed in Table 3 in descending order of frequency with which they appeared.

The importance of hoarseness as a symptom has frequently been pointed out. On the other hand, the frequency with which nonspecific symptoms, such as cough, decreased general vigor, dysphagia, and dyspnea, occur in this disease, should emphasize the importance of routine laryngoscopic examinations for all patients over 40 years of age.

NEGATIVE FIRST BIOPSY

Fourteen per cent of our patients who proved eventually to have cancer of the larynx had negative first biopsies. Histological diagnoses included hemangioma, benign polyp, papilloma, hyperkeratosis, and chronic inflammation. One patient had proved cervical metastases at the time the negative laryngeal biopsy was reported. In the majority of such cases a second biopsy, repeated very shortly

TABLE 3.—*Presenting Symptoms in Order of Frequency*

Symptom	Cases	
	No.	%
Hoarseness	189	81
Cough	101	43
Decreased general vigor (including weight loss, anorexia)	93	40
Dysphagia	92	39
Dyspnea	72	31
Pain	66	28
Sore throat	51	22
Neck swelling	20	9
Stridor	5	2
Regurgitation	1	1

after the first, revealed cancer at the same site. It is a frustrating experience to take what one considers an adequate biopsy of a lesion which is clinically cancer and get a negative report. Nine of the patients mentioned above required three or more biopsies before a tissue diagnosis was made. On the other hand, the gross appearance of a laryngeal lesion may be quite misleading. This was dramatically demonstrated in the case of an 81-year-old man who had what appeared to be cancer of the larynx on which biopsy was performed eight times over a period of four years, revealing epithelial hyperplasia, chronic inflammation, and granulation tissue on the various reports. The ninth section showed highly anaplastic carcinoma. This tumor could hardly have been present for four years, particularly since the patient made an excellent recovery after laryngectomy and postoperative irradiation and is living and well at present, 23 months later. Suspicious-looking lesions must be followed closely.

DURATION OF SYMPTOMS IN PROGNOSIS

Of the 52 patients who survived five years or more, 40% were treated within 6 months and a total of 81% within the first 18 months after the onset of symptoms.

METASTATIC LESIONS

Metastatic lesions present at the time of diagnosis included cervical lymph node and pulmonary metastases. Four patients had lung metastases at the time of diagnosis; five had lung and cervical metastases, and 109, or 46%, had palpable cervical, submental, or supraclavicular nodes. Of these nodes, 35, or 32%, were proved cancerous by biopsy. In the present series, of the 109 patients having palpable nodes recorded by at least one observer at the time of diagnosis, 31% had nodes which were proved histologically to be metastatic. When these proved nodes were palpable, surgery was withheld in most cases and other treatment given, depending upon the extent of the disease. Occasionally, however, such nodes were overlooked by most observers, only to be demonstrated as metastases at laryngectomy a few days later. In such cases, postoperative irradiation, either by deep x-rays or by radon seeds, was the treatment most frequently employed. Of these 30 cases with proved cancerous cervical nodes, there were 3 with five-year survivals, or a salvage rate of 10%. Our present feeling about the management of such cases is discussed further under "Neck Dissection."

TABLE 4.—*Grade of Malignancy and Five-Year Survival*

Grade	Number of Cases Followed 5 Yr.	5-Yr. Survival	% 5-Yr. Survival
I	11	4	36
I or II	1	..	0
II	51	21	41 (40 for I and II)
II and III	1	1	100
III	19	2	11
IV	0	0	.. (11 for III and IV)

HISTOLOGICAL DIAGNOSIS

Ninety-five per cent of the tumors in this series were epidermoid carcinoma. It is interesting to note that of the 13 malignant tumors other than epidermoid carcinoma, 5 appeared in women. Or, to put it another way, 27% of the female patients had lesions other than epidermoid carcinoma, as compared with 4% of the male patients.

GRADING

Until 1946, lesions on which biopsies were performed at this hospital were frequently but not always graded by the department of pathology according to Broders' classification. About 1946, or even before, it became increasingly apparent to the pathologists here that a single grade was inadequate and frequently incorrect. Different portions of the same lesion, and even of the same slide, not infrequently differed in degrees of malignancy, ranging all the way from I to IV. Grading was therefore abandoned, and pathological reports at this institution consist at present of carefully detailed description of the gross and microscopic findings, in addition to the diagnosis, which is occasionally accompanied by some descriptive phrase, such as "well differentiated," "highly anaplastic," "intraepithelial," etc. It is then entirely the clinician's responsibility to evaluate the prognosis of the patient and decide upon treatment in view of his knowledge of the location, extent, and histo-

logical nature of the cancer as well as the patient's general condition. The value of the figures in Table 4 is open to serious question, since less than 50% of the cases followed for five years and only 53% of the five-year survivals were graded.

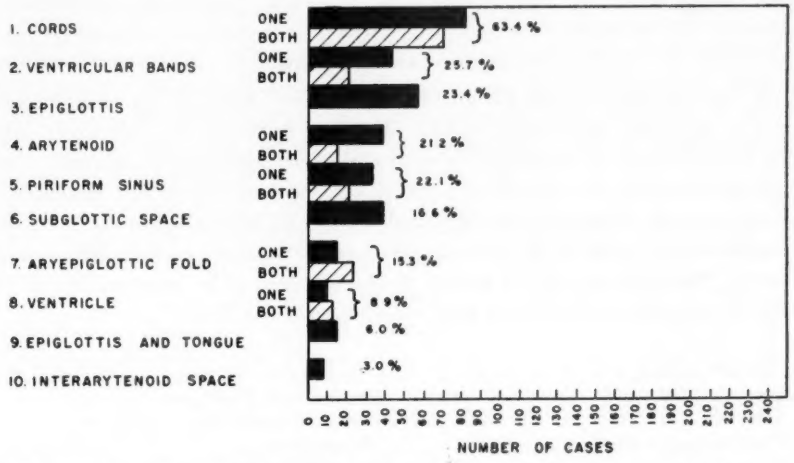

Chart 2.—Frequency of involvement of specific sites at the time of diagnosis in 235 cases of cancer of the larynx.

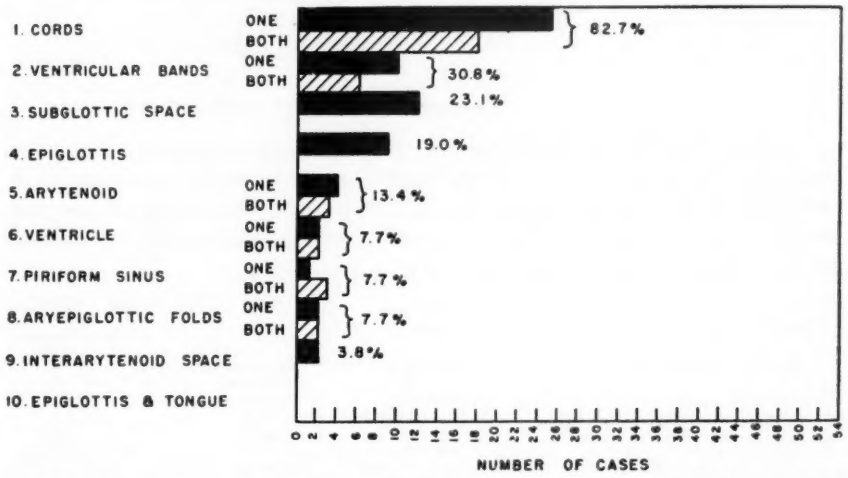

Chart 3.—Sites involved in 52 cases of cancer of the larynx with survival of five years or longer.

Nevertheless, it can readily be seen in Table 4 that the five-year survivals in cases of grade I and II lesions were almost four times as high as those found in cases of grade III and IV lesions. These limited figures, then, would seem to attest to the value of Broders' classification in determining prognosis.

LOCATION OF LESIONS AT TIME OF DIAGNOSIS

A modified form of Orton's [6] classification was used in the present study (Chart 2). The true cords, ventricular bands, epiglottis, and arytenoid and piriform sinuses, in that order, were the sites most frequently involved in this series. Lesions of the arytenoid and the piriform sinus were most frequently unilateral.

LOCATION AND PROGNOSIS

In the five-year survival group, 83% had lesions involving one or both cords (37% of these had cord lesions alone); 31% had lesions of the ventricular band, 23% had extension to the subglottic space, 19% had lesion of the epiglottis (in 2 of the 9 cases there was no other site, and all but one were treated with irradiation), and one or both arytenoids were involved in 13% of these cases. It is interesting to compare our results in all forms of treatment with those of Orton in his evaluation of surgical treatment of lesions of specific sites. The two lists of sites in order of prognosis are presented here for comparison.

Orton [6] (Surgery)	New Haven Hospital (All Modes of Treatment)
1. True and false cords	1. True and false cords
2. Epiglottis	2. Subglottic space
3. Epiglottis and tongue	3. Epiglottis
4. Arytenoid	4. Arytenoid
5. Ventricle	5. Ventricle
6. Subglottic space	6. Piriform sinus
7. Piriform sinus	7. Aryepiglottic fold
8. Aryepiglottic fold	8. Epiglottis and tongue

TREATMENT

1. *Local Excision, Intralaryngeal Extirpation.*—Local excision was most often done as a biopsy procedure for benign-appearing lesions which turned out to be malignant on histological examination. Usually, further therapy was advised, but in four cases it was refused. These four cases provided an opportunity to evaluate excision alone and are summarized below:

Case	Location	Grade	Palpable Nodes
1	Epiglottis	Malignant amelanotic melanoma	0
2	Epiglottis	Undifferentiated (no grade)	0
3	Right cord (polyp)	Grade II	0
4	Both true and false cords (multiple papillomata)	Grade III	0

This very small series of cases suggests that differentiated polypoid lesions limited to one or both cords might be amenable to such conservative therapy, since

6. Orton, H. B.: Symposium: Carcinoma of the Larynx; II. Treatment of Extensive Carcinoma of the Larynx, Laryngoscope 61:496-510 (June) 1951.

three of the four patients survived more than five years. LeJeune recently outlined the indications for intralaryngeal extirpation.[7]

2. *Laryngofissure.*—Before 1947, six patients were treated by laryngofissure alone. Of these, 4, or 67%, had five-year cures, and 2 are still alive. All had differentiated epidermoid carcinomas, and one of the four cured patients had palpable nodes at the time of diagnosis. Compared with these six laryngofissures recorded between 1921 and 1947, nine have already been done in the years since 1947. Seven patients lived more than one year, and six are still living. Five had lesions limited to one cord; three had lesions involving both cords, and in two the lesions involved one cord and the anterior commissure. All were epidermoid carcinoma; none was graded.

TABLE 5.—*Results of Laryngectomy, 1921-1946 (Fixty-Six Cases)*

How Used	Cases, No.	Five-Year Survivals No.	Five-Year Survivals %
Laryngectomy alone	27	14	52
Laryngectomy combined, total	29	12	42
Laryngectomy preceded by x-ray	8	4	50
Laryngectomy followed by x-rays *	12	4	33
Multiple combined	9	4	44
Laryngectomy combined with x-rays, neck dissection, radon	2	1	50
X-rays, radical neck dissection	1	..	0
X-rays, radium	1	..	0
Neck dissection, radon	1	1	100
Hemilaryngectomy, x-rays	1	1	100
X-rays, local excision	3	1	33
3. Total laryngectomies	56	26	46

* In six of these irradiation was done because of recurrence.

3. *Hemilaryngectomy.*—Of the four patients treated by hemilaryngectomy, all were treated before 1947. Three patients lived at least five years after the operation. In these three cases the growth involved one cord in two and a single cord and the anterior commissure in one. All the growths were epidermoid carcinomas.

4. *Laryngectomy.*—Laryngectomy was the major form of therapy for 56 patients treated before 1947; 26 of the 56, or 46%, survived five years or more. Twenty-seven had laryngectomy alone; five-year survival in this group was 52%. Twenty-nine patients had other forms of therapy in addition to laryngectomy; 12, or 42%, of these had five-year survivals.

Our technique is that of wide-field resection, including ribbon muscles, the mid-third or more of the hyoid bone with the preepiglottic space and the epiglottis.

5. *Neck Dissection.*—Four patients followed five years or more were treated with some type of neck dissection in conjunction with other surgical, and occasionally radiological, therapy; all had palpable nodes, and three were proved histologically. Most of these dissections were not "radical," as the term is used today. Two of these patients survived five years or more. The problem of neck dissection

7. Le Jeune, F.: Symposium: Carcinoma of the Larynx; I. Surgical Treatment of Early Cancer of the Larynx, Laryngoscope **61**:488-495 (June) 1951.

in this disease has recently been reviewed by Ogura [8] and by Martin.[9] Our indications for neck dissection at present are in close accord with those outlined by Martin.[9] We have not, however, used needle biopsy, since we feel that suspicious-looking nodes should be excised and examined histologically. If the node is proved malignant, the area of excision is included in the neck dissection.

6. *Radiation Therapy.*—Radiotherapy was the sole form of treatment in 71 cases followed for five or more years; in 16 of these, or 23%, five-year cures were obtained. Our patients received high-voltage radiation of a calculated tumor

TABLE 6.—*Incidence of Involved Sites in Sixteen Cases Cured by X-Ray*

Site	Cases	
	No.	%
Cords	9	55
Ventricular bands	7	44
Arytenoids	6	37
Aryepiglottic folds	3	19
Piriform sinus	3	19
Epiglottis	3	19
Interarytenoid space	2	12
Ventricle	2	12
Subglottic	2	12

TABLE 7.—*Results of Treatment 1921-1946*

Treatment	Patients				
	Total No.	No. Surviving			5-Yr. Survival, %
		1 Yr.	5 Yr.	5 Yr.	
Surgical *	70	13	21	36	51
Local excision	4	..	1	3	75
Laryngofissure	6	2	..	4	67
Hemilaryngectomy	4	1	..	3†	75
Laryngectomy	56	10	20	26	46
Irradiation ‡	71	35	20	16	22

* Twenty-nine patients had combined surgical and radiation therapy.
† One of these patients had a recurrence, which was treated by laryngectomy.
‡ Only patients treated by radiation alone are included in this group.

dose (range 3,000 to 7,000 r) with or without local use of radium or radon. All lesions were epidermoid carcinoma except one spindle cell sarcoma. The relative frequency with which specific sites were involved in the 16 cases with cure is listed in Table 6.

In the majority of cases, the cords, ventricular bands, and arytenoids were involved. However, sites such as the piriform sinus, epiglottis, and aryepiglottic folds also responded well. It is interesting to note that of the 16 surviving patients 12 had edema during or after irradiation, belying the impression held in some quarters that the development of edema during treatment indicates a poor prog-

8. Ogura, J. S.: Laryngectomy and Radical Neck Dissection for Carcinoma of the Larynx, Tr. Am. Acad. Ophth., pp. 786-808, Nov.-Dec. 1951.

9. Martin H.; Del Valle, B.; Erlich, H., and Cahan, W. G.: Neck Dissection, Cancer 4:441-499 (May) 1951.

nosis. In evaluating this 23% cure rate, it should be pointed out radiotherapy has usually been reserved for patients with very extensive "nonsurgical" lesions or for patients whose associated organic disease makes them poor candidates for surgery. Best results were obtained in those patients with either differentiated or undifferentiated lesions, involving the cords, ventricular bands, and arytenoids. Lesions of the aryepiglottic fold, piriform sinus, epiglottis, interarytenoid space, ventricle, and subglottic space were next in order. Table 7 summarizes the results obtained at New Haven Hospital with various modes of treatment.

MORBIDITY

In studying the complications of treatment we have been particularly impressed by two factors: (1) the decreasing incidence of postoperative infection with the use of antibiotics, and (2) the importance of eliminating oral sepsis and extracting carious teeth before operation in minimizing wound infection and fistulas.

SURGICAL MORTALITY

The surgical mortality in this series was 6%. Of the 11 deaths, 7 were due to postoperative pneumonia.

SUMMARY AND CONCLUSIONS

Two hundred thirty-five cases of proved cancer of the larynx observed at New Haven Hospital between 1921 and 1951 have been studied.

Males outnumbered females in a ratio of 12:1. The prognosis differed for the two sexes; 50% of the women survived five years or more, whereas only 28% of the men lived so long.

Age at onset of symptoms in 65% of the cases was between 50 and 70 years with women tending to get the disease at an earlier age than men.

Thirteen per cent of the patients had family histories of malignancy. Thirty-four per cent admitted to excessive alcohol intake. Forty-three per cent were known to smoke or chew to excess. Twenty-three per cent of all patients were engaged in metal working occupations.

Thirty per cent had x-ray diagnoses of tuberculosis, about one-third of these having active lesions. Evidence of syphilitic infection was present in 6% of cases. The possible role of infection in carcinogenesis is discussed.

Septal deviation with obstruction appeared in 31% of patients; its effect and that of mouth breathing on respiratory epithelium are considered.

Hoarseness and cough in 81 and 43% of cases, respectively, were the most frequent presenting symptoms. Unilateral painless neck swelling was the first symptom in a surprisingly large number of cases, 6%.

Forty-seven per cent of patients sought medical care within six months of onset; 73%, within the first year. Fourteen per cent of patients had a first biopsy which was negative for cancer. Forty per cent of patients had laryngeal edema noted at the first examination.

Ninety-five per cent of lesions were epidermoid carcinoma. Twenty-seven per cent of the female patients in this series had histologic types other than epidermoid carcinoma, as compared with 4% of males.

Eight per cent of the patients had no treatment of any kind; 95% received therapy in some form. Thirty-one per cent were treated by surgery, 48% by radiotherapy, and 21% by a combination of surgery and irradiation. The over-all five-year survival rate in the cases in which treatment was given was 34.2%. Intralaryngeal extirpation was performed in four cases, with three five-year cures.

Laryngofissure resulted in a 67% five-year cure rate. Hemilaryngectomy produced a 75% five-year survival in a small series of cases.

The five-year survival rate after laryngectomy ranged between 42 and 52%, depending upon whether or not additional therapy was required. The majority of patients on whom laryngectomy was successful had differentiated lesions involving one or both cords, with or without fixation, with extension to the subglottic space, ventricular bands, ventricle, epiglottis or all of these. Results of neck dissection as an adjunct to other therapy were promising in a limited series of cases. Radiotherapy alone yielded 22.5% five-year survival. The relationship of anatomical location to prognosis is discussed. The development of edema during irradiation had no adverse effect on the cure rate.

The preoperative extraction of carious teeth and other dental prophylaxis are important in the prevention of postoperative complications.

BILATERAL POLYPOID GRANULOMA OF THE LARYNX FOLLOWING ENDOTRACHEAL ANESTHESIA

Report of a Case

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ATHENS, GREECE

THE EXTENSIVE use of endotracheal anesthesia has brought to light some pathological alterations of the vocal cords following injuries which had formerly been observed but which, owing to the limited use of this kind of anesthesia, had not drawn due attention.

Recently published works give no description of bilateral polypoid granuloma of the larynx following endotracheal anesthesia, thus not being up-to-date with regard to this specific clinical feature, its etiology, laryngoscopic picture, and histology. More emphasis must be given to the expression "specific clinical feature," since the lesion is a part of the nosological entity simple chronic hyperplastic laryngitis with polypoid elaboration which develops on a substratum of simple chronic laryngitis or follows an operation.

This paper concerns exclusively the so-called laryngeal granuloma (of no specific origin) following endotracheal anesthesia, or "inflammatory polypoid granuloma of the larynx" following endotracheal anesthesia to which attention was first drawn by R. J. Clausen, in Great Britain, in 1932.[1]

Use of the term "bilateral polypoid granuloma of the larynx following endotracheal anesthesia," if universal, would greatly lessen the confusion caused by use of different terms for the same disease.

Since Clausen's report the number of observations has increased, and I have been able to gather from the literature 33 reports exclusive of my own. In 1951 Flagg[2] reported 101 cases of inflammatory polypoid granuloma of the larynx following endotracheal anesthesia; one might ask whether all cases of this complication are reported or even whether cases already published are included in this number.

It is well known that the first cause of this lesion is introduction of the tube (direct trauma), particularly when catheterization of the trachea is performed blindly or through a glottis constricted by spasm or when a catheter of larger

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From the Otorhinolaryngological Clinic of "Asclipiion Voulas" of the Greek Red Cross, Dr. Ioannovich Lecturer and Director.

1. Clausen, R. J.: Unusual Sequela of Tracheal Intubation, Proc. Roy. Soc. Med. **25**:1507-1508 (Aug.) 1932.

2. Flagg, P. J.: Endotracheal Inhalation Anesthesia: Special Reference to Postoperative Reactions and Suggestions for Their Elimination, Laryngoscope **61**:818-831 (Aug.) 1951; Incidence and Control of Trauma Accompanying Endotracheal Anesthesia: A Survey Based on the Response of 150 Leading Laryngologists and My Experience of 25 Years in the Use of the Method, Arch. Otolaryng. **53**:439-445 (April) 1951.

caliber than the glottis is used. The second cause consists of factors favoring the necrosis resulting from friction or pressure (indirect trauma). Thorough relaxation of the vocal cords is therefore recommended. These factors probably were the cause in my case, although thorough relaxation of the vocal cords occurred before introduction of the tube. When an endotracheal tube is used with a small rubber bag at its free end to obstruct the trachea, it may slip backward, and the bag then presses the vocal cords. A third type of cause is such predisposing factors as a recent laryngeal-pharyngeal-nasal and paranasal-sinus infection, overuse of the voice after endotracheal anesthesia with unskillful maneuvers, imperfect asepsis, and mucous secretions. To eliminate the infection factor and determine the morphologic and functional state of the larynx, it is advisable that endotracheal anesthesia be preceded by a thorough examination of ears, nose, and larynx and by indirect laryngoscopy, the usually performed direct laryngologic examination being a more radical maneuver in a patient with an inflamed substratum.

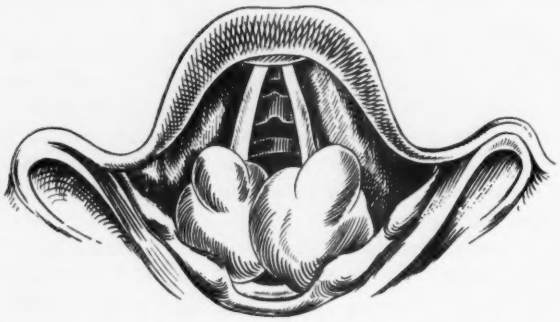

Fig. 1.—Indirect laryngoscopy diagrams of larynx showing a bilateral granuloma on posterior third of each cord (vocal process) during respiration.

This complication may occur even when the anesthetist is experienced (New and Devine [3]). A fourth cause is Magill's catheter curve, according to some investigators,[4] who recommend its modification to a double curve.

Published cases indicate a predominance of the complication in females and an age range from 32 to 60.

Pathological examination shows the polypoid granuloma to be unilateral, or bilateral, pedunculated or sessile, and clearly and firmly localized on the mucous membrane of the posterior third of the vocal cords by the vocal process of the arytenoid cartilages of the larynx. This site of predilection is explained most probably by the marked friction or pressure which occurs between the cartilaginous substratum and the catheter, thus giving rise to an ulceration and hemorrhagic infiltration of the epithelium. There is more commonly a unilateral growth (ratio, 20:34) than a bilateral one (ratio, 14:34). My case was one of bilateral localization (Figs. 1 and 2). Histological examination reveals newly formed granular connective tissue.

3. New, G. B., and Devine, D. D.: Contact Ulcer Granuloma, Ann. Otol. Rhin. & Laryng. **58**:548-558 (June) 1949.

4. Peimer, R., and Feuerstein, S. S.: Laryngeal Granuloma Following Intratracheal Anesthesia: A Report of 4 Cases, Laryngoscope **61**:165-171 (Feb.) 1951.

Clinically, the subjective findings are hoarseness of voice, impaired swallowing, and slight pain, and the objective findings, cough with mucosanguineous expectoration and slight pain on external pressure of the larynx. Laryngoscopy reveals inflammatory post-traumatic edema of various degrees, localized chiefly on the vocal processes of the arytenoid cartilages of the larynx. Moreover, one may observe hemorrhages at different sites on the vocal cords. These are the initial symptoms. This clinical picture is ordinarily not observed, and there is perfect healing. In some, happily rarer cases the alterations become permanent, assuming the characteristics of "granuloma." Hoarseness progresses to aphony. Within one to seven months mild breathlessness may have advanced to dyspnea and then cyanosis, which constitute the advanced symptoms. At laryngoscopy one may

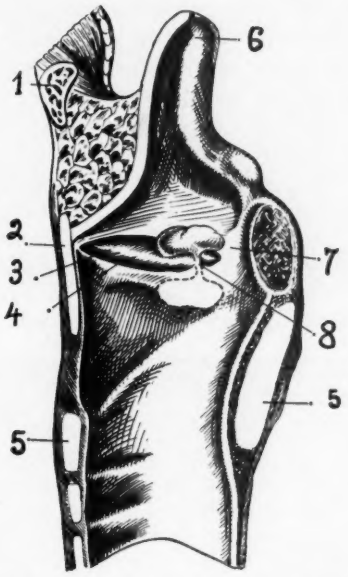

Fig. 2.—*1*, hyoid bone; *2*, thyroid cartilage; *3*, laryngeal ventricle; *4*, true vocal cord; *5*, cricoid cartilage; *6*, epiglottis; *7*, polypoid granuloma; *8*, vocal process to which the pedunculated granuloma is attached. Dotted lines show granuloma directed toward subglottic space during respiration. Thus examination of larynx during phonation is recommended in order to avoid errors.

observe at the posterior third of the vocal cords a unilateral or bilateral growth, sometimes pedunculated and sometimes sessile, localized on the vocal process of the arytenoid cartilages or at its proximity, with complete mobility of one or both altered vocal cords.

REVIEW OF THE LITERATURE

Cohen [5] in 1938 reported a case of unilateral polypoid granuloma of the larynx, which developed two months after endotracheal anesthesia applied blindly by the

5. Cohen, M.: Tumor of Vocal Cord Following Nasal Endotracheal Anesthesia, Brit. M. J. **1**:283-284 (Feb. 5) 1938.

nasal route after repeated attempts. This procedure was one of several causes of the growth.

Lundy [6] in 1942 admitted that trauma is caused by introduction of the tube but did not accept this as the only cause of the growths, owing to the fact that such granulomas also develop without intubation. He did not consider the length of time the tube was in place to be significant.

Flagg [7] in 1944 stated that friction of the tube on the mucous membrane lining the vocal cords, with secondary infection, is due to use of a dry tube during spasm of the vocal cords.

McLaurin [8] in 1948 recommended that no tube wider than a 32 F. Magill catheter should be used endotracheally and that the tube should be introduced by direct laryngoscopy.

New and Devine [3] in 1949 expressed some doubts as to the etiologic factor, the kind and the duration of the operation, and the method and difficulty of introducing the catheter. It was their opinion that this complication is possibly not so rare as it was believed on the basis of the few published cases.

Gould [9] in 1935 reported a case in which the patient, a woman, three months after endotracheal anesthesia had attacks of dyspnea with cyanosis. The catheter had been introduced blindly but without difficulty. After removal of the polypoid granuloma another tumor developed, removal of which was followed by permanent healing.

Smiley [10] in 1940 reported a case of polypoid granuloma of the larynx which developed ten days after endotracheal anesthesia. It was evidenced by expectoration of mucosanguineous material and, after one month, difficulty in breathing. The patient refused operation.

Kearney [11] in 1946 reported a similar case associated with dyspnea.

REPORT OF CASE

A woman aged 21, treated in the fourth clinic of the "Asclipiion Voulas" of the Greek Red Cross for tuberculous spondylitis of the dorsal part of the vertebral column, underwent a typical Albee operation on May 9, 1951, with endotracheal anesthesia. The operation lasted 21 minutes.

The night before operation the patient was given 0.2 gm. of pentobarbital (Nembutal) sodium orally. The day of the operation she received 0.2 gm. of pentobarbital sodium at 6 a.m. and 0.010 gm. of morphine sulfate with 0.0005 gm. of scopolamine hydrobromide subcutaneously at 8 a.m. Anesthesia was induced at 9:20. Before the intubation 6 mg. of curare (tubocurarine chloride) and 500 mg. of thiopental (Pentothal) sodium (of the 2.5% solution, that is, 20 cc.) was administered intravenously. Seven minutes after the induction of anesthesia the intubation was performed by direct laryngoscopy. Since direct laryngoscopy showed slight laryngospasm

6. Lundy, J. S.: Clinical Anesthesia: A Manual of Clinical Anesthesiology, Philadelphia, W. B. Saunders Company, 1942.

7. Flagg, P. J.: The Art of Anesthesia, Ed. 7, Philadelphia, J. B. Lippincott Company, 1944, p. 1378.

8. McLaurin, J. W.: Bilateral Granuloma of the Larynx Following Endotracheal Anesthesia, abstracted, Quart. Rev. Otorhinolaryng. & Broncho-Esophag. **7**:202 (June) 1948.

9. Gould, R. B.: Laryngeal Granuloma Following Intratracheal Intubation, Brit. M. J. **2**:449-500 (Sept. 14) 1935.

10. Smiley, W. A.: Polypoid Granuloma of the Larynx Following Endo-Tracheal Anesthesia, Ann. Otol. Rhin. & Laryng. **49**:556-558 (June) 1940.

11. Kearney, H. L.: Bilateral Granuloma of the Larynx Following Intratracheal Anesthesia, Ann. Otol. Rhin. & Laryng. **55**:185-187 (March) 1946.

and the vocal cords situated paramesially, a 1% tetracaine (Pontocaine) hydrochloride solution was applied by spray to eliminate the reflexes, which finally regressed. The vocal cords being in complete abduction after relaxation of the spasm, a 34 F. Magill-Forreger endotracheal catheter was introduced easily by direct laryngoscopy. At the end of the operation the catheter was with-

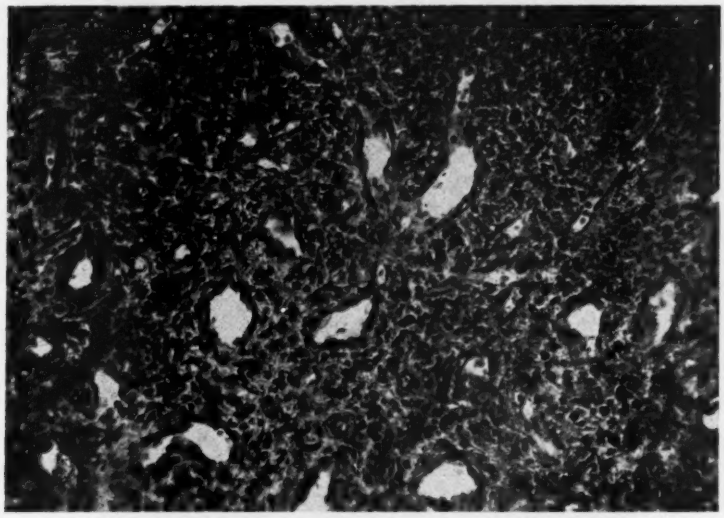

Fig. 3.—Inflammatory granuloma with newly formed blood vessels.

Fig. 4.—Vessels are seen grossly in this section of polypoid granuloma.

drawn smoothly. On the second postoperative day the patient complained of hoarseness. This lasted for three months and varied in intensity. Indirect laryngoscopy showed circumscribed movable growths superimposed on and occupying the posterior thirds of both vocal cords (Figs. 1 and 2). Examination of the lungs performed by the clinician did not show any abnormality.

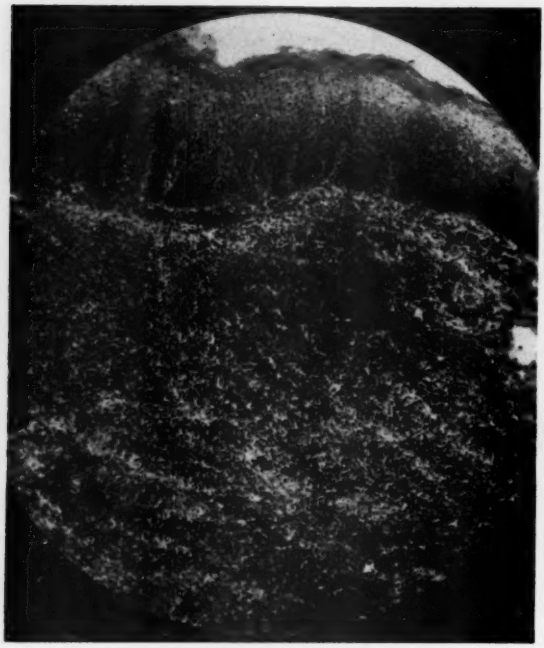

Fig. 5.—Section of polypoid granuloma showing that surface is lined in places only by squamous stratified epithelium.

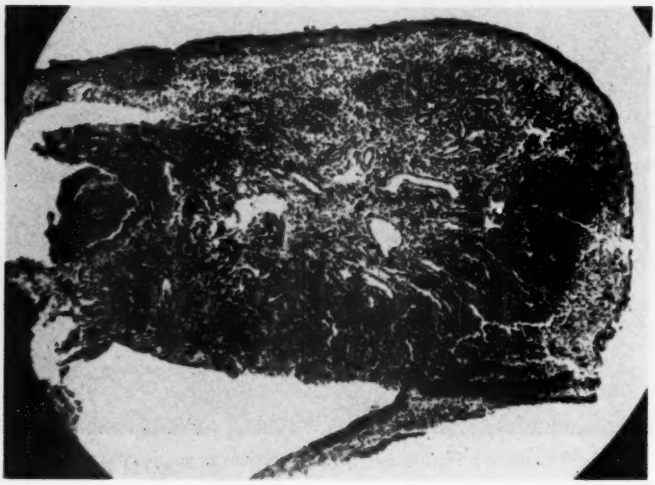

Fig. 6.—Section of polypoid granuloma showing that surface is mostly ulcerated.

Examinations of the sputum for Koch bacilli and Wassermann and Kahn tests of the blood were negative.

Endolaryngeal removal of the growths by indirect laryngoscopy was easy. The pedunculated and highly movable growth of the left vocal cord was removed first in a single maneuver without hemorrhage. Then the less-movable sessile growth of the right vocal cord was partly removed in two maneuvers. This growth was larger than the left one, and visibility of the operative field was hindered by hemorrhage due to a highly vascularized area. The remainder was removed later. Thereafter the patient presented a normal laryngoscopic picture. Histological examinations showed that the processes made up an inflammatory granuloma (inflammatory polypoid processes) (Figs. 3, 4, 5, and 6,).

COMMENT

Evolution of the clinical picture depends on the laryngeal stenosis caused by the growth: aphonia—dyspnea—cyanosis. Rarely, the granuloma is expelled by cough after detachment of its delicate pedicle, as happened in the two published cases of Clausen [1] and Ezz-El-Din El-Samma.[12] In about one-third of the cases, one may observe a new growth following an imperfectly removed granuloma, owing to the rich vascularization. New growth has been observed twice and, more rarely three times.

Treatment consists in the endolaryngeal removal of the growth, particularly when it has become pedunculated. It is to be emphasized that the removed granuloma must be histologically examined; there may be concealed pathology, as has been observed by New and Devine.[3] Removal is followed by the usual management. The traumatic surface must be reexamined for new growth.

CONCLUSIONS

Precautions should be taken to avoid injuries during endotracheal anesthesia. Such injuries are rare now but may be more frequent in the future, owing to the increasing use of this kind of anesthesia.

It is wise, before inducing endotracheal anesthesia, to have the patient examined by indirect laryngoscopy, as is recommended before direct laryngoscopy and tracheobronchoscopy. Chronic inflammations of the larynx, favoring the development of similar changes after the anesthesia, are a temporary contraindication.

It is equally wise to introduce the endotracheal catheter by direct laryngoscopy rather than blindly, and when the vocal cords are in perfect abduction, as it is well known that the barbiturates intravenously administered give rise to laryngospasm. To avoid spasm, experienced laryngologists recommend surface anesthesia with a spray of 2% tetracaine hydrochloride.

After endotracheal anesthesia the larynx must be examined, and the patient is instructed to avoid coughing, intense phonation, and frequent speech.

I do not mean on the basis of the medical literature and my own case to dampen enthusiasm for endotracheal anesthesia. The number of cases of injury published in detail is small—about 30—as against many thousands of cases in which endotracheal anesthesia has been used.

12. Ezz El-Din El-Samma: Polypoid Granuloma of the Larynx Following Endotracheal Anesthesia, J. Roy. Egyptian M. A. **33**:912-917, 1950.

FACTORS AFFECTING PSYCHOLOGIC ADJUSTMENT IN THE LARYNGECTOMIZED PATIENT

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LARYNGEAL surgery, particularly laryngectomy, has assumed a large place in the practice of otolaryngology during the past 10 or 15 years. During the last five years, at least 400 articles have appeared in the medical literature on the treatment of laryngeal carcinoma; this number testifies to the widespread interest in the subject and to the fact that surgical treatment is now generally available to the victims of this disease. From the medical and surgical standpoint, the results of laryngectomy are favorable for the survival of the patients and the cure of the lesions, but the fact that the patients recover satisfactorily does not cancel out the striking disability they have to face after the operation; that is, the loss of normal speech. The capacity for articulate speech is the most important feature distinguishing man from the animals. When a man can no longer talk, he feels himself less a man, and this fact in itself causes significant emotional disturbance.

As all otolarynogologists realize, the psychic trauma connected with laryngectomy is severe and is one of the most serious problems in the management of patients with carcinoma of the larynx. Hence it seems surprising that so little attention has been given to this particular problem in the current literature. The only publications I was able to find which deal with psychologic factors specifically were two papers by Greene,[1] which appeared in 1947 and were based on his experiences with patients at the National Hospital for Speech Disorder, in New York City.

In his paper on "Laryngectomy and Its Psychologic Implications," Greene emphasized that most of the efforts directed toward the treatment of patients with cancer of the larynx have been focused on the mechanics of surgical technic and on the mechanics of esophageal speech and of artificial voice boxes. Nevertheless, the psychic trauma incident to laryngectomy is easily as serious as the physical trauma itself and should never be underestimated in dealing with the patient after operation.

From a detailed study of the psychologic factors in 70 patients and from observations on another 200 patients seen in the Postlaryngectomy Clinic over a period of several years, Greene made the following recommendations for dealing with laryngectomized patients:

Presented as a candidate's thesis in partial fulfillment of the requirements for membership in the American Laryngological, Rhinological and Otological Society, Inc.

1. Greene, J. S.: Laryngectomy and Its Psychologic Implications, New York J. Med. **47:** 53-56 (Jan. 1) 1947; Treatment of Disorders of Speech, Postgrad. Med. **1:**44-50 (Jan.) 1947.

1. The patient should be told frankly before the operation that the larynx must be removed and exactly what this will mean in terms of losing his voice and curtailing his activities. The patient is in better condition physically and psychologically to stand the shock before than immediately after operation. Patients who received a frank simple explanation recovered their equilibrium more quickly than those who were unprepared and discovered the full extent of their misfortune only after the operation.

2. Before the operation, the patient should be visited by a patient who has lost his larynx and has a good speaking voice.

3. The same person should call on the patient immediately after the operation, because then the patient is at the nadir of his depression.

4. As soon as possible, the patient should receive voice training and this should be carried on away from the hospital in which the operation took place.

5. The rehabilitation program should embody psychologic as well as reeducational measures; for this purpose group therapy is recommended.

My own interest in the psychologic problems encountered by laryngectomized patients has been growing throughout the past several years, as I have kept in close touch with most of the patients on whom I have performed the operation and have taken an active interest in the group-therapy club in Cleveland. My interest was brought to a more definite focus early this year when one of my patients, whose operation was performed about five years ago, complained that he had experienced a period of depression about his condition after having previously made a good adjustment. From this case and one or two others that had come to my notice, it seemed possible that there might be a late psychologic trauma connected with laryngectomy, which might have been overlooked.

I decided to make a more detailed study of the matter, to see what conclusions might be drawn about this and other factors concerned in the patient's adjustment to his handicap. To collect the necessary data, the following questionnaire was formulated and sent to approximately 200 laryngectomized persons, including many operated upon by my colleagues and some from other cities:

QUESTIONNAIRE

1. What was the date of your operation?

2. Do you feel that you had adequate explanation and preparation for your operation?

3. Did you receive preoperative esophageal voice training? How many days?

4. Did you see the moving picture "New Voices"? How did it affect you?

5. Have you any suggestions that would help us dispel fear and instil confidence in laryngectomized patients that they will be able to talk?

6. On what day after operation did your spirits fall the lowest?

7. Did anyone who had had the operation visit you at this time? If so, was your confidence increased?

8. How long was it before you felt that your esophageal voice was adequate for you to return to your work?

9. Do you have frequent periods of self-pity? Resentment?

10. Do you become annoyed when other people ask you, "What is wrong with your voice?"

11. Have you been a member of a "Group Therapy" club composed of laryngectomized individuals? Have you remained active in teaching others to talk?

12. Are you now carrying on the same work you did before operation? If not, what is your present job?

13. What bothers you most at the present time?

14. Remarks: Your own ideas, thoughts, suggestions.

TABLE 1.—*Information from Eight Patients Operated on Less Than One Year Before (1952)*

Preparation before operation: None, 3; inadequate, 1; adequate, 3; questionable answer, 1

Preoperative esophageal voice training: None, 6; minimal, 2

Moving picture: Yes, 4; no, 4. Reaction: Favorable, 3; indifferent, 1

Time of most serious depression: No answer, 2; no postoperative depression, 2; 1st postoperative day, 1; 6th postoperative day, 1; 10th postoperative day, 1; 1 or 2 mo. after operation, 1

Hospital visitor: Yes, 5; no, 3. Effect: Favorable, 3; unfavorable, 1; indifferent, 1

Esophageal voice: Not talking or speech inadequate for return to work, 5; able to resume light work 20 days after operation, 1; housewife, resumed duties, 1; return to work with aid of electrolarynx, 1

Self-pity: No answer, 2; yes, 2; no, 4

Resentment: No answer, 2; yes, 3; no, 3

Annoyance with questions about voice: No answer, 4; yes, 1; no, 3

Membership in group-therapy club: No answer, 2; yes, 3; no, 3

Participation in teaching: No answer, 3; willing, but still learning, 3; no, 2

Occupation: Same as before, 2 (1 housewife); not same, but working, 1; not working yet, 5

Chief complaint: No answer, 1; none, 2; economic insecurity, 1; trachea tube, 1; worry over health and inability to talk, 1; noise when talking, 1; inability to talk and trachea tube, 1

TABLE 2.—*Information from Twelve Patients Operated upon One Year or More Before (1951)*

Preparation before operation: None, 3; adequate, 7; inadequate, 2

Preoperative esophageal voice training: None, 11; minimal, 1

Moving picture: Yes, 6; no, 6. Reactions: Favorable, 1; unfavorable, 2; no answer, 3

Time of most serious depression: Never depressed, 3; 3rd day, 1; 5th day, 1; just before leaving hospital, 1; after return home, 1; 1st 4 mo., 1; 1st 6 mo., 1; does not recall, 1; no answer, 1

Hospital visitor: Yes, 8; no, 2; no answer, 2. Reaction: Favorable, 7; unfavorable, 1

Esophageal voice: Not talking but returned to work, 3; cannot work, 1; returned to work in 3 wk., 1; returned to work in 1 mo., 4; in 9 wk., 1; in 3 mo., 1; in 6 mo., 1

Self-pity: No, 8; Yes, 4

Resentment: No, 6; yes, 4; no answer, 2

Annoyance with questions about voice: No answer, 1; no, 9; yes, 2

Membership in group-therapy club: Yes, 5; no, 7

Participation in teaching: Yes, 1; no, 5; willing, but unable to speak, 3; no answer, 3

Occupation: Same as before, 9; partly, 1; no, 2

Chief complaint: None, 2; provoked at those who think he cannot hear, 2; trying to be heard in noisy places, 3; inability to talk, 4; lack of voice volume, 1; mucus from neck opening, 1

Comment: 2 patients use a mechanical larynx; 1, who had been doing well, died approximately 1 yr. after operation from virus infection

TABLE 3.—*Information from Ten Patients Operated upon Two Years or More Previously*

Preparation before operation: Inadequate, 3; adequate, 7

Preoperative esophageal voice training: None, 6; minimal, 2; adequate, 2

Moving picture: Yes, 6; no, 4. Reaction: Favorable, 4; equivocal, 2

Time of most serious depression: No depression, 4; preoperative, when told about operation, 1; 3rd day after operation, 1; 4th and 5th days after operation, 1; 1 wk. after operation, 2; 1st 3 mo., 1

Hospital visitor: Yes, 3; no, 4; no, but patient called on person who had had operation, 1; no answer, 2. Effect of visit: Favorable, 2; unfavorable, 1

Esophageal voice: Few weeks after x-ray treatment, 1; about 6 wk., 2; about 6 mo., 1; not yet adequate, 1; about 1 yr. (difficulty getting job), 1; no answer, 2; not working, 3

Self-pity: No answer, 2; no, 6; yes, 2

Resentment: No answer, 3; no, 5; yes, 2

Annoyance with questions about voice: No, 6; yes, 4

Membership in group-therapy club: Yes, 5; no, 5

Participation in teaching: No, 5; yes, 2; no answer, 3

Occupation: Same as before, 6 (1 housewife); not same, but working, 3; approximately same, 1

Chief complaint: None, 1; insufficient voice volume, 2; inability to talk, 3; talking where noisy, 1; mucus in throat and coughing, 2

Comment: 1 patient using an artificial larynx

TABLE 4.—*Information from Nine Patients Operated upon Three or More Years Before (1949)*

Preparation before operation: Inadequate, 3; adequate, 6

Preoperative esophageal voice training: None, 7; minimal, 1; adequate, 1

Moving picture: Yes, 5; no, 4. Reaction: Favorable, 4; unfavorable, 1

Time of most serious depression: None, 1; no answer, 1; cannot recall, 1; immediately after operation, 2; 3rd or 4th day, 2; 5th day, 1; 2nd wk., 1

Hospital visitor: Yes, 5; no, 4. Reaction: Favorable, 5

Esophageal voice and return to work: No answer, 1; to work without talking in 2 wk., 1; to work without talking in 6 mo., 1; 2 mo., 1; 3 mo., 3; 4 mo., 1

Self-pity: Yes, 4; no, 4; no answer, 1

Resentment: Yes, none; No, 6; no answer, 3

Annoyance with questions about voice: Yes, none; no, 9

Membership in group-therapy club: Yes, 5; no, 4

Participation in teaching others: Yes, 4; no, 3; willing, but no opportunity, 1; no answer, 1

Occupation: Same as before, 4; not same, but working, 1; not working, 3; approximately same job, 1

Chief complaints: Talking difficulty, 3; pneumonia every year, 1; lack of voice volume with noise, 1; none, 1; occasional colds, 1; discharge of mucus, 1

Comment: 1 patient died 6 mo. after operation; 1 had been a superintendent in his plant but had to do ordinary factory work (with same company) after operation

TABLE 5.—*Information from Seven Patients Operated upon Four or More Years Before (1948)*

Preparation before operation: Inadequate, 2; adequate, 5

Preoperative esophageal voice training: None, 6; adequate, 1

Moving picture: Yes, 1; no, 6. Reaction: Favorable, 1

Time of most serious depression: None, 3; 3 or 4 days after operation, 2; day of operation, 2

Hospital visitor: Yes, 2; no, 5. Reaction: Favorable, 2

Esophageal voice and return to work: 5 wk., 1; 2 mo., 1; 3 mo. (to work but not talking), 1; 8 mo., 1; 1 yr., 1; not working, 1; working, with artificial larynx, 1

Self-pity: Yes, 1; no, 5; no answer, 1

Resentment: Yes, 2; no, 4; no answer, 1

Annoyance with questions about voice: Yes, 1; no, 6

Membership in group-therapy club: Yes, 2; no, 5

Participation in teaching speech: Yes, 3; no, 4

Occupation: Same as before, 2; not same, but working, 2; not working, 3

Chief complaints: Coughing spells, 2; inability to talk, 1; lack of voice volume, 1; mucus discharge, 1; "Can't swim," 1; none, 1

TABLE 6.—*Information from Nine Patients Operated upon Five to Ten Years Before (1943-1947)*

Preparation before operation: Inadequate, 1; adequate, 7; questionable, 1

Preoperative esophageal voice training: None, 6; minimal, 3; adequate, 1

Moving picture: Yes, 3; no, 5; no answer, 1. Reaction: Favorable, 1; not stated, 2

Time of most serious depression: None, 3; before operation, 1; 2 wk. after operation, 3; 1 mo. after operation, 1; no answer, 1

Hospital visitor: Yes, 7; no, 1; no answer, 1. Reaction: Favorable, 5; unfavorable, 1; indifferent, 1

Esophageal voice and return to work: To work immediately, but voice inadequate for 6 mo., 1; to work in 6 wk., but 2 yr. without voice, 1; without voice, use of electrolarynx, 3; 8 wk., 1; 4 mo., 1; 4 wk., 1

Self-pity: No, 9; yes, none

Resentment: No, 7; yes, 2

Annoyance with questions about voice: Yes, 1; no, 8

Membership in group-therapy club: Yes, but not now, 2; yes, 2; no, 4; no answer, 1

Participation in teaching speech: Yes, 1; no, 7; no answer, 1

Occupation: Same as before, 7; not working, 2

Chief complaint: Mucus discharge, 2; colds in winter, 1; hot weather, 1; lack of volume and effort in talking, 1; no speech class in home town, 1; "Getting old," 1; none, 1

Comment: Note that 3 patients use an artificial larynx

The data thus collected have been augmented by many personal interviews and some correspondence with these and other patients, and all the facts accumulated have been interpreted in the light of impressions gained from continuous observation of many of these patients over a considerable period, to form the basis of the present report.

FACTS GAINED FROM THE PATIENTS' QUESTIONNAIRES

The answers received on the questionnaires have been grouped chronologically according to the time of operation, and the results appear in Tables 1 to 7. Specific comments, suggestions, and remarks were, as a rule omitted from the tabulations.

The first question, as to the time of operation, furnished the basis for classifying and grouping the cases. In Table 1 are presented the data from 8 patients whose operations were performed within the last year; in Table 2, information from 12 operated upon during 1951; in Table 3, data from 10 operated upon during 1950; in Table 4, information from 9 operated upon during 1949; in Table 5,

TABLE 7.—*Information from Six Patients Operated upon Ten to Twenty-Two Years Before 1931-1942*

Preparation before operation: Inadequate, 3; adequate, 3
Preoperative esophageal voice training: None, 6
Moving picture: Yes (later), 1; no, 5
Time of most serious depression: Never, 3; 1 wk. after operation, 2; 4 mo. after operation, 1
Hospital visitor: Yes, 4; no, 2. Reaction: Favorable, 4
Esophageal voice and return to work: Retired at time of operation, 1; voice still inadequate, 1; returned to work without voice in 2 mo., 2; returned to work in 4 wk., 1; returned to work in 10 yr., 1
Self-pity, Yes, none; no, 5; no answer, 1
Resentment: Yes, 1; no, 5
Annoyance with questions about voice: Yes, 1; no, 5
Membership in group-therapy club: Yes, 1; no, 5
Participation in teaching: Yes, 3; no, 2; no answer, 1
Occupation: Same as before, 2; not same, but working, 1; not working, 3
Chief complaint: Mucus in tube, 3; none, 3

data from 7 operated upon during 1948; in Table 6, information from 9 whose operations were performed from 1943 to 1947, and in Table 7, data from 6 operated upon from 1931 to 1942, i. e. from 10 to 20 or more years ago. Four additional questionnaires were returned, but these did not give the date of operation, and so they were excluded from the tables.

The next three questions give significant information as to the preoperative preparation received by laryngectomy patients, and the answers indicate that much improvement is possible in this matter. Those of us who perform laryngectomies should take note of the large number of patients who feel that they were inadequately prepared for the psychologic shock of losing their voice. Approximately one-third of the patients represented in this study felt that they did not have adequate preparation by the physician before the operation. A much larger number, approximately three-fourths, had no preoperative instruction or practice in belching to aid them in the development of esophageal speech after operation. Of those who received some preoperative training, only five felt that this was adequate. The moving picture "New Voices" was seen by approximately half the patients operated upon since 1949, either before or immediately after operation. The reactions of those who saw this film were somewhat mixed. The majority

felt that it was helpful, but there were numerous patients who felt that it discouraged rather than encouraged them, and a few were indifferent in their reaction.

As for the time of the most serious depression, the questionnaires indicate that this varies considerably, although it seems evident that the majority of patients experience this low point within the first week or two after operation. A good many of those who were operated on some years ago commented that they were depressed for four or six months after the operation. These comments are interesting in view of the fact that the answers from patients operated upon within the past year indicate that their spirits are much lower, as a general rule, than those who were laryngectomized a longer time ago. The inability to talk during the first weeks and months and the economic insecurity caused by the inability to return to work because of the speech difficulty make the early period difficult. Patients who apparently make the best adjustment during this period are those who display the strongest determination to learn esophageal speech.

The questionnaires and the comments sent by many of the patients indicate that one of the principal factors in building morale both before and after operation is the opportunity to talk with a laryngectomized person who has good esophageal speech. Such an interview builds confidence as nothing else can, for it proves to the patient that someone else has achieved satisfactory rehabilitation and there is no reason why he should not make the effort to do so. The questionnaires indicate, however, that much more can be done in this connection, for 18 patients, nearly a third of the entire group, reported that they did not have a visit from a laryngectomized patient in the hospital. Those who did receive such a visit, as a rule, reacted favorably, although six patients stated that the visitor depressed them because he did not talk well enough to be impressive and the effect was discouraging rather than otherwise.

The answer to Question 8 indicates that there is a wide variation in the time of returning to work and in the acquisition of speech that permits the holding of a job. The return to work is obviously dependent on many factors, including the patient's will and need to work, the type of job he has held or is capable of holding, his age, his general philosophic outlook, whether or not he is emotionally a retiring or an aggressive person, as well as his ability to learn esophageal speech. A few patients returned to work as early as two weeks after the operation, and numerous others returned within the first three months, before they had mastered the art of esophageal speech. A few found it necessary, because of their work, to resort to the use of a mechanical larynx or the electrolarynx but later were able to talk with an esophageal voice.

Question 9 deserves some comment, because it is my impression that the negative answers which predominate do not give the true picture of the patients' psychologic attitudes in regard to self-pity and resentment. More were willing to admit that they felt resentful than that they felt sorry for themselves. Several of those whose operations were performed years before admitted that they had felt self-pity during the period immediately after the operation, but said that after learning to talk they no longer felt that emotion. Eleven patients answered the question about self-pity affirmatively; many who denied this emotion revealed by their special comments or complaints that the denial was not significant. The reason this question was included in the questionnaire was that it had been my impression in dealing with laryngectomized patients that self-pity and resentment

at their fate were among the main obstacles to successful rehabilitation. It is apparent from clinical experience and also from the material in the questionnaires that those who make the best adjustments to the handicap of losing the larynx are those who have some religious or other philosophy that sustains them. Conversely, those who have the most difficult time, even in learning esophageal speech, are those who are more self-centered. Those who are anxious to help others are the ones who get along best themselves. This is a principle which has been amply demonstrated in the group-therapy work exemplified by such organizations as Alcoholics Anonymous. Group-therapy clubs for laryngectomized persons, if properly organized and administered, can perform a similar service for their members.

The answers to Question 11 indicate that many laryngectomized persons have not enjoyed the advantage of the group therapy offered by such organizations. In some instances this is because they live in smaller communities, where there is no opportunity for such activity, but in far too many instances the failure to join an organization is due to selfishness or inertia. Only 23 of the 61 patients, slightly more than one-third, indicated that they were active in a club for laryngectomized persons. Two or three additional ones indicated that they had once belonged but had dropped out. Only 14 persons indicated that they were active in helping others learn to talk. Besides these, seven of the patients operated upon more recently expressed a willingness to teach others as soon as they could learn themselves.

The answers to Question 12, regarding employment, are, on the whole, encouraging. When it is remembered that many patients with laryngeal cancer are in the older age groups, which would mean that they would be retired anyway, the number of patients who are not working is not excessive. Half the patients (31) reported that they were engaged in the same occupation as before the operation. Eleven additional patients were working but not at the same job they had before operation. In most of these instances, the change of work had been necessitated by speech difficulty or inability. Of the 18 patients who reported that they are not employed, 5 were operated upon during the last year and expect to get jobs as soon as they can improve their speech.

Of the complaints listed in Question 13, the outstanding difficulties have to do with speech, either inability to talk, lack of voice volume, or too much noise while talking. In connection with the speech difficulty, several patients complained that they become annoyed by the typical reaction of others, who scream at them as if they were deaf. Another major complaint is the annoyance of mucus in the tracheal tube, which is exacerbated by intercurrent infections. Coughing and mucus discharge were mentioned by numerous patients as the chief causes of their discomfort. Economic insecurity and worry over health were mentioned only by patients who were operated upon during the current year, another indication that the first year apparently is the hardest.

COMMENT

Although the facts elicted by this questionnaire did not reveal any trend toward late psychic trauma, as had been suspected as a possibility before the study was made, the information stresses in a vivid way the difficult emotional and psychologic adjustment required of the laryngectomized patient. Furthermore, the facts, as set forth by the patients, along with their comments and suggestions,

indicate that there are many ways in which otolaryngologists are not fulfilling their responsibility toward these patients and that numerous practices might be improved to add to the patients' welfare and peace of mind. Responsibility devolves on the laryngologic surgeon when he carries out the procedure of separating the individual from his normal means of communication, and he—the surgeon—should follow the patient routinely through all the phases of preoperative preparation and postoperative care, so that he may be on the alert to detect any evidence of recurrence, become cognizant of the patient's emotional and psychologic difficulties, and contribute all in his power toward alleviating them.

To comprehend these difficulties, it must be remembered that the patient with carcinoma of the larynx is faced first of all with the terrible fact that his disease is malignant; this fact arouses all the fears of cancer and of its recurrence that his experiences and observations have created. He next realizes that to save his life his voice must be sacrificed and that he will not be able to communicate normally after the larynx is removed. However well this situation is explained to him, the complete impact of the experience is difficult for him to anticipate, and for many patients there is no proper realization beforehand of the meaning of this hard fact. If he receives no proper explanation of the change in his life and activities that will result from the operation, he is naturally bitter and resentful toward the surgeon when he is faced with the shock of it after surgery has been performed. The questionnaires and interviews with patients in connection with this study revealed that a lack of proper preparation is the chief cause of resentment on the part of laryngectomized patients.

Another fact elicited in connection with the preoperative handling of patients with carcinoma of the larynx is that some patients feel resentful toward the family physician because the malignant nature of the condition was not promptly recognized and when they were finally referred to the specialist they were rushed into surgery, sometimes the same day, with no preparation whatsoever as to the actual nature of the treatment. This situation is not the fault of the otolaryngologists, of course, but it points up the fact that there must be no remission of effort to inform the general physician and the public of the significance of the symptom of chronic hoarseness.

The speed with which some patients with carcinoma of the larynx have been rushed into operation is illustrated by the following comment from one of them: "I was told at 5 p. m. on Monday and operated on Tuesday morning at 8:30 a. m. Had I known the results I would have refused the operation. In my case death would have been so much better." This comment is typical of several in similar instances. The patient obviously not only was told nothing of the effects of laryngectomy, but his preparation also was inadequate in that he felt perhaps that the operation was unnecessary. It is evident that often the patient fails to understand that the operation on the larynx is a lifesaving measure.

If the questionnaires make it obvious that in some instances the laryngologist has failed to prepare and instruct the patient adequately before operation, they also indicate that many surgeons do an excellent job of reassuring their patients and instilling confidence in them preoperatively. For example, one patient wrote: "I think a doctor has a lot to do to dispel fear. My doctor handled my case so expertly and with a personality that expelled all fear. Another thing that was a

big help was the confidence I had in him. His personality and the method with which he handles his patients is wonderful.

Instruction of the family is a part of the laryngologist's responsibility, too, and many patients mentioned the help that understanding and encouragement on the part of the family had been to them. One patient commented that the family of the laryngectomized person should receive some preparation as to how to react toward him. One patient, a minister, suggested that the laryngectomized person should not be scolded or discouraged by members of his family unless there are sure signs of laziness in his or her effort.

As to preoperative esophageal voice training, the answers from patients show that this is by no means generally practiced. There is some disparity of opinion regarding the efficacy of preoperative training, which accounts for some of this neglect. It is the opinion of my colleagues and myself on the basis of experience at our hospital, that a few lessons before operation, to explain the mechanism of esophageal voice production and to give the patient some practice in controlled belching, hasten the process of learning esophageal speech after operation. Hence, whenever possible the patient should have the benefit of this preoperative training. Although some patients reported that they did not react favorably to the moving picture "New Voices," nevertheless the general reaction is a positive one, and, unless there is some individual psychoneurotic disability to contraindicate it, the picture should be shown to all patients who are about to undergo laryngectomy, to prove to them that it is possible to learn to talk. Visits from previously laryngectomized patients before operation, as well as after operation when the patient is most depressed, are excellent morale builders and probably the most effective stimulus toward the effort of acquiring esophageal speech. However, all the patients who discussed this factor emphasized that the person who calls on the patient about to undergo laryngectomy should be one whose appearance is neat and who has really mastered the art of talking through the esophagus. When poor speakers pay these visits, the hospital patient is likely to be more depressed than encouraged.

A comment by a member of a group-therapy club brought out a point that we otolaryngologists may sometimes overlook. This man made a plea that physicians cooperate with club members in referral of patients and in arranging the time of visits. To quote his own words: "Doctors should make it possible at all times to cooperate with members who are trying to assist their patients. Members' time is just as valuable to them as the doctor's time, and he should assist them in all ways. They don't want thanks. They just want cooperation."

In the struggle toward adjustment in the weeks and months immediately after the operation, the surgeon should keep in close touch with the newly laryngectomized patient, so as to be ready to offer helpful suggestions and advice in his struggle to learn to talk and to rehabilitate himself. The number of patients who reported on the questionnaire that they never learned anything at all about esophageal speech from their surgeon but happened on the information from some outside source is surprising. Or, if they did discover from the surgeon that there was a way to learn a new method of talking, they did not receive concrete information from him as to where to go or whom to consult in order to be taught. This

indifferent attitude toward speech rehabilitation is not the general rule today, however, for in all the large cities, otolaryngologists cooperate with group-therapy clubs and try to stimulate patients toward learning. This type of interest must be continued and expanded if the best interests of the laryngectomized patients are to be served.

When the patient does make the effort to learn to talk, it behooves the surgeon to realize that there are many factors which may influence his progress favorably or adversely. In the first place, there is great individual variation, both from the physical and from the psychologic standpoint in the ability to master esophageal speech. Some patients can learn the trick of belching and can control the stomach muscles easily, and others never can learn. For those for whom belching is difficult, the use of carbonated beverages is often a help at first, but some patients learn to depend on these and claim they never can produce sounds without ginger ale or beer.

The most important factor in influencing the progress in learning speech postoperatively is the emotional make-up of the individual. Patients with intelligence and the will to learn are willing to endure the struggle and to perform the practice required. Confidence, reassurance, and understanding help are what the patient needs from his surgeon and from his family. And, most of all, he needs the help of those who have had to learn esophageal speech themselves, for they make the best teachers. In the matter of helpful advice to the laryngectomized patient, the surgeon must base this on an adequate appraisal of the patient's emotional and psychologic make-up. Some patients are never able to learn esophageal speech, and if, after an adequate trial, it becomes apparent that the patient will not be able to learn, it is perhaps better for the surgeon to recommend that the effort be abandoned, to improve both the patient's morale and that of his family. In such instances and in certain cases in which economic necessity demands an early return to work, mechanical-larynx instruments may be helpful. In the case of some patients who are learning or have learned esophageal speech, these instruments may serve as an aid at times of stress or when the patient has a cold.

The surgeon should inform himself as thoroughly as possible regarding the problems encountered in the learning of esophageal speech, so that he can help his patients avoid some of the more obvious pitfalls. As already mentioned, it is important, from both the psychologic and the mechanical standpoint, that some instruction be given in the art of belching before the operation. Afterwards, it is important that the patient be encouraged to try to talk immediately and that he never resort to whispering or the use of pad and pencil. It is important to know, too, that certain individual factors present special difficulties, such as previous speech difficulties or speaking with a foreign accent. The more the physician understands about the production of esophageal speech, the better the advice and reassurance he can offer the patient struggling to learn. The surgeon, in some instances, should work in close cooperation with the speech teacher in the handling of special problems, and he should always, so far as it is possible for him to do so, display an interest in the group-therapy clubs.

A complaint of some of the patients was that a fee is charged in most instances for lessons in esophageal speech. Many feel that these lessons should be free to all laryngectomized patients and suggest that some program be worked out

through the Cancer Society to make free lessons possible. This is a problem to which laryngologists should direct some thought; the solution will provide satisfaction to all concerned. There are professional teachers, themselves laryngectomized persons, who devote all their time to this work and naturally have to have remuneration for it. Whether payment should come from organizations or from the individual pupils is the question at issue, and the answer probably cannot be a general one.

In the learning of esophageal speech, most patients benefit by group therapy. For this reason clubs composed of laryngectomized persons are now functoning in most of the larger communities, and recently a national organization was formed. Although many members give devoted service to this project and do a wonderful work in encouraging patients in the hospital and in helping them to learn to talk, there is considerable criticism of these groups. It is said by some people that when laryngectomized persons get together there is too much talk about physical symptoms and difficulties and that this talk may prove depressing. The fact is that many persons attend for a while and then drop out.

As has been noted with reference to the complaints cited in the questionnaire, the difficulty that disturbs the laryngectomized person the most, aside from efforts to speak, is use of the tracheal tube, with discharge of mucous and coughing, especially when there is an acute respiratory disorder. These problems to some become a mere matter of routine, and to others they are disagreeable obstacles that can never quite be surmounted. Although some of this difference is due to psychologic attitude, some of it is caused by individual variations in the physical and mechanical factors involved. The majority of patients operated upon 10 years or more ago still have to wear a tracheotomy tube. With improvement in surgical technic, the number has been reduced to about one-half the patients operated on. Among the last 25 laryngectomies I have performed, only one patient has required a permanent tracheal tube. The majority of patients should be decannulated as soon as possible, that is, within the first three months. The Hays Martin plastic procedure for the repair of the tracheal stump has proved satisfactory and apparently could be much more widely employed among otolaryngologists. My experience has shown that elimination of the tracheal tube is a big factor in psychologic adjustment, since most patients are much happier with this prime complaint out of the way.

SUMMARY AND CONCLUSIONS

The answers to a questionnaire received from 65 laryngectomized patients have been tabulated and discussed, with the object of emphasizing the many factors which influence the progress and welfare of the patient from the psychologic and emotional standpoint.

The facts elicted by the questionnaire have indicated many ways in which laryngologists might improve the care of these patients by more careful attention to the details of preoperative preparation, encouragement in the use of esophageal speech, reassurance and helpful suggestions in regard to emotional problems, and, through continuous improvement in surgical technic, elimination of some of the difficulties connected with the wearing of the tracheal tube. The patient should be reassured and protected at times of acute colds or upper respiratory inflammation,

for these represent periods of difficulty and possibly also of danger. The patient should be relieved of the fear of recurrence, so far as this is possible, for this is a major obstacle to well-being in some cases.

A proper selection of cases is important in the practice of laryngeal surgery. Some patients are of such a maladjusted, immature emotional make-up that they may never be able to make the adjustment required by loss of the larynx. In their cases, roentgen therapy may be preferable to surgery. It behooves the surgeon to make a careful estimate of the emotional make-up before operation is undertaken, for often more depends on the psychologic characteristics of the patient than on the technical procedures used in treatment. The best results always are obtained in the mature, out-going person who wants to get well and who wants to live.

Dr. Julius W. McCall provided me with a complete list of his laryngectomized patients and the dates of operation.

EFFECTS OF CORTISONE IN CHRONIC OTITIS MEDIA

A Preliminary Report

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THE WIDESPREAD use of corticotropin (ACTH) and cortisone in various fields and the particular benefits which ophthalmologists [1] have witnessed, together with reports of the response of otosclerosis,[2] led to the belief that chronic otitis media might be another condition in which to try cortisone.

The primary aim of the studies to be reported was to determine the effects of cortisone (Cortone) drops in chronic otitis media, and my attention was directed to whether cortisone would alter or suppress the secretion, heal the perforation, destroy granulation tissue, or produce any other response.

Fourteen patients with chronic otitis media were treated with cortisone ear drops. Most had previously received local and parenteral medication of some sort intermittently without any response. All the patients were seen for the first time during May and June, 1952, and repeated examinations were done two to four months after medication for the purpose of noting any effects or recurrences. The patients who did not respond to cortisone were later given antibiotics [3] locally and parenterally.

PROCEDURE

No cultures were attempted in these studies because of certain difficulties. However, in a group of seven patients, precautions were taken to minimize to some extent the virulence of infection, particularly that due to Pseudomonas aeruginosa; the canal was irrigated with half-strength vinegar [4] solution [5] after being cleaned and was dried after irrigation with sterile cotton pledgets.

The other seven patients were not given vinegar irrigations in order to eliminate the possibility that the irrigations might have been sufficient to stop the discharge.

From the Department of Eye, Ear, Nose, and Throat, University of Santo Tomás College of Medicine.

1. Smith, R. W., Jr., and Steffensen, E. H.: ACTH and Cortisone in the Treatment of Ocular Disease, New England J. Med. **245**:972-977 (Dec. 20) 1951.

2. Angeluscheff, Z.: Otosclerosis and ACTH: Preliminary Report, A. M. A. Arch. Otolaryng. **54**:200 (Aug.) 1951.

3. Penicillin G sodium, 200,000 units, and dihydrostreptomycin sulfate, 1 Gm., were mixed with 20 cc. of isotonic sodium chloride solution. Three drops of the mixture was instilled in the ear canal four times a day, and Dicrysticin (Squibb), a mixture containing 300,000 units of penicillin procaine, 100,000 units of penicillin G potassium and 0.5 gm. of dihydrostreptomycin sulfate in each dose, was injected daily intramuscularly.

4. Native vinegar, boiled and filtered, with equal parts of sterile distilled water, was used.

5. Ochs, I. L.: Use of Vinegar as an Antibiotic in the Treatment of Chronic Middle Ear Disease, A. M. A. Arch. Otolaryng. **52**:935-941 (Dec.) 1950.

Cortisone [6] (25 mg. per cubic centimeter diluted with 6 cc. of isotonic sodium chloride solution) was used as ear drops during waking hours. Patients were advised to instill the solution into their ears, 1 drop every two hours for the first two days and 1 drop every four hours for another four days, and then to report for observation. If there was little or no improvement medication was continued for another 4 days, making a total of 10 days. Patients in both groups were observed after the 10 days of local cortisone medication for another 10 days at 5 day intervals, and results were noted.

Patients were cautioned not to insert cotton pledgets into the canal for fear of reinfection, but they were allowed to wipe away excess solution that might drip to the auricle. When the patient reported the ear was cleaned with sterile cotton pledgets to remove cortisone crystals that had dried in the canal and to facilitate examination of the drum.

REPORT OF CASES

Group 1: Ears Irrigated with Half-Strength Vinegar Solution Previous to Use of Cortisone Ear Drops

Case 1.—K. L. Y., a Chinese girl aged 14, was seen on May 5, 1952, because of intermittent left aural discharge since the age of 12. The secretion was fetid, abundant, and purulent. The drum membrane had a central perforation. After 10 days of cortisone medication the secretion became scanty and mucoid. Antibiotics were later used, and the secretion dried after six days of medication. No recurrence was reported after September, 1952.

Case 2.—J. M., a Filipino man aged 33, was seen May 14, 1952, with a right aural discharge of 15 months' duration. The secretion was purulent, and the drum membrane had a marginal perforation. On the 10th day of treatment the secretion was less abundant and was mucoid. The ear dried with antibiotics in 10 days. The patient was again seen Sept. 16, almost four months later. There had been no recurrence.

Case 3.—M. K., a Filipino man aged 22, was seen may 20, 1952, because of a bilateral purulent aural discharge which had been present since he was 12 years old. The drum membrane in the right ear had a marginal perforation, and that in the left ear had a tubal perforation. The right mastoid region was tender on palpation. The discharge decreased and became mucoid after treatment. After two weeks of observation both ears were still wet. The treatment was therefore changed to use of antibiotics. After five days both ears were dry. The patient reported Aug. 18, two months after treatment, with a recurrence in the right ear.

Case 4.—C. B. L,. a Chinese boy aged 16, appeared for examination May 26, 1952, because of deafness and purulent discharge in the right ear of 11 months' duration. On examination the canal was seen to be filled with a purulent material, and after cleaning a tubal perforation was observed. After use of cortisone for six days the secretion was mucoid, and on the 20th day (June 16) the ear was dry. The patient was seen three months later (Sept. 22), and had had no recurrence.

Case 5.—J. L. D., a Chinese girl aged 12, appeared for examination May 28, 1952, because of a bilateral purulent aural discharge present since she was 8. Both drums had a central perforation. No appreciable change was noted in either ear during the course of treatment and after another two weeks of observation, but the ears dried after six days of antibiotic treatment. When seen in September, the patient had had no recurrence.

Case 6.—C. F., a Filipino boy aged 7, was seen May 28, 1952, because of a purulent discharge present in the right ear since he was 4 years old. The drum had a marginal perforation in the posterior-superior quadrant, with some granulation tissue. With cortisone treatment the secretion decreased and became mucoid. The granulation tissue became smaller than it had

6. Cortisone Acetate (Cortone Acetate Merck) 25 mg. per cubic centimeter was used.

been on the first day. The ear dried after 12 days of antibiotic treatment. The parents brought the child back because of conjunctivitis in September, three months after treatment, and the ear was still dry.

Case 7.—P. Z., a Filipino woman aged 21, was seen May 29, 1952, because of intermittent purulent aural discharge on the left, present since she was 14. The canal was filled with secretion. After cleaning the drum membrane showed a marginal perforation in the posterior-superior quadrant. A mass was present in the perforation. The patient had a history of mastoid pain and tenderness two years before, which disappeared after she took sulfonamides. The secretion became scanty and mucoid on the 10th day of treatment, and after 15 days the mass was smaller. It took 10 days of antibiotic treatment to dry the ear. The patient has not been seen since.

Group 2: Ears Not Previously Irrigated

Case 1.—J. F., a Chinese youth aged 19, was seen May 7, 1952, because of a purulent discharge in both ears present since he was 10 years old. Both drum membranes had a marginal perforation with granulation tissue. There was a history of high fever and mastoid pain a year before, at which time he was given several injections of penicillin. He was relieved temporarily, but three weeks later the secretion was again abundant. Ten days after local cortisone therapy the secretion was scanty and mucoid, but the ears had not dried after two weeks of observation. I suggested an x-ray of the mastoids, but the patient did not come back.

Case 2.—M. S. L., a Chinese man aged 21, appeared for examination May 15, 1952, because of deafness and a fetid, purulent discharge in the left ear of one year's duration. On examination the external auditory canal was seen to be congested, the secretion was greenish, an a tubal perforation was present. Cortisone was used locally for 10 consecutive days and the secretion turned mucoid, congestion of the canal disappeared, and fetor was checked. After 10 days of observation the ear was still wet. After two weeks, therefore, antibiotics were given, and it took another eight days to dry the ear (June 27). No recurrence had been reported by September, about three months after treatment.

Case 3.—P. S., a Filipino youth aged 18, was seen May 17, 1952, because of a purulent discharge in the left ear, with diminished hearing, of two years' duration. After the canal was cleaned a central perforation was seen. Local treatment with cortisone was started at once, and after six days the secretion was scanty. The ear dried after the 10th day of medication. When the patient was seen in September more than four months later, there had been no recurrence.

Case 4.—F. C., a Chinese man aged 38, was seen May 20, 1952, because of a purulent discharge of both ears of four years' duration. He also complained of a recent hearing impairment. The drum membrane had a central perforation with granulation tissue on both sides. The canal was congested. After 10 days of cortisone treatment there was only an alteration in the character of the secretion; with antibiotic medication the right ear was dry in 6 days and the left on the 11th day. The granulatioin tissue was smaller than at the first examination. No recurrence was reported up to September.

Case 5.—C. C., a Filipino boy aged 15, appeared for examination May 26, 1952, because of impaired hearing and a fetid discharge of the left ear of three years' duration. The drum had a marginal perforation in the posterior-inferior quadrant. The ear did not dry with cortisone, but the secretion became mucoid and scanty and fetor disappeared. The patient was given antibiotics, and the discharge stopped after eight days. He was not seen afterward.

Case 6.—L. A., a Filipino woman aged 21, came for examination May 29, 1952, because of a discharge and deafness in the left ear due to swimming one year before. The secretion was purulent, and the perforation was marginal. After cortisone treatment the secretion stopped. The patient reported monthly for observation, and the ear was still dry at the last examination, Sept. 29.

Case 7.—A. C. L., a Chinese boy aged 12, was brought for examination by his father June 6, 1952, because of a fetid purulent discharge in the left ear. This had started a year before. The canal was congested, and the drum membrane had a central perforation. No alteration in the character of the secretion was observed during the cortisone treatment. The ear responded to antibiotics in four days. Up to October, no recurrence was reported.

It is interesting to note that cortisone influenced the course of chronic otitis media in both groups, except in Case 5 of Group 1 and Case 7 of Group 2, in which no alteration in the character of the secretion or any other response was noted and in both of which the patient responded satisfactorily later to antibiotics. Patient 4 of Group 1 had had otorrhea for 11 months and had a tubal perforation. Patients 3 and 6 of Group 2, who had had otorrhea for two years with a central perforation and for one year with a marginal perforation, respectively, responded to cortisone ear drops without other medication.

In the remaining nine cases of the two groups, aside from the alteration in the character of the secretion, the following observations were noted. The inflammatory reaction diminished. Congestion of the external auditory canal from constant bathing with the secretion became less. The red margins that are frequently seen in perforated drums owing to constant irritation by the discharge were not apparent. Epidermal invasion in marginal perforations, granulation tissue, and polyps seen in some of the ears showed no evidence of progress; instead, the granulation tissue appeared smaller than it had at the first examination. The drum membranes showed evidence of regaining luster, and the epithelial debris and calcareous deposits commonly seen in pathologic drum membranes were absent. Fetor was checked.

All patients who were treated with antibiotics after cortisone ear drops failed to stop the discharge reported satisfactory results except Patient 3 of Group 1, who had a mastoid extension in the right ear and reported a recurrence after two months. No follow-up was made in Case 7 of Group 1 and Case 5 of Group 2. Patient 1 of Group 2 did not receive antibiotic treatment. I did not observe any perforation of the drum membrane that healed or closed, nor was hearing improvement claimed by any patient. No untoward reactions were noted with cortisone ear drops; however, some patients in Group 1 complained of earache with half-strength vinegar irrigation.

COMMENT

Dr. Joseph Lee Hollander's [7] statements that "cortisone and ACTH help many conditions but cure none," and "extreme caution is urged in their use" were in my mind when I first thought of trying cortisone for chronic otitis media. The reports in the literature and our free access to antibiotics are encouraging to a continuance of studies of the hormones, particularly when an infecting agent is acting as the stimulus.

This study has further demonstrated the reported actions of cortisone in reducing or diminishing inflammatory reactions,[8] inhibiting fibroplasia, causing involution of lymphoid tissue,[9] and reducing allergic reactions.[10]

In chronic otitis media the constant bathing of the middle ear and canal with the secretion is enough to induce the inflammatory and allergic conditions observed. The failure of perforations to close and the diminution in size of granulations and polyps are accounted for by the nature of the action of cortisone.

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7. Hollander, J. L.: Practical Problems in the Use of Cortisone and ACTH, abstracted, A. M. A. Arch. Otolaryng. **55**:520 (April) 1952.

8. Lepri, G.: Studies on Cortisone in Ophthalmology, Am. J. Ophth. **35**:935-946 (July) 1952.

9. The Therapeutic Use of Cortone, Rahway, N. J., Merck & Co., Inc., 1950.

10. Allergic Conditions, Cortone News, No. 7, Rahway, N. J., Merck & Co., Inc., 1952.

There is no doubt that in otitis media purulenta, whether it is of the acute or chronic type, a bacterial infection exists and the tissues respond to this particular stress by an inflammatory reaction. Cortisone, because of its ability to neutralize the effects of stress, favors the return of the tissue to its normal state, and the normal resistance to bacterial invasion is thus regained, so that whatever micro-organism is present in the middle ear can be adequately taken care of by the body.

Patients in whom only an alteration in the secretion was observed responded to antibiotics after the cortisone treatment. It was probably because of a diminution in the inflammatory reaction in the middle ear that the antibiotics later could act directly on the infecting agent. As Dr. Andres Cruz[11] has stated, a lytic effect exerted by cortisone on the serofibrinous secretion, similar to the observed action of streptokinase, exposes the micro-organism to the antibiotic action of the antibiotics used. The reported recurrence after treatment in cases of mastoid involvement is probably due to a flaring up of a latent infection.

SUMMARY

Cortisone in a 1:6 dilution was used as ear drops in a series of 14 cases of chronic otitis media. Only three patients responded to treatment. Those who failed to respond were later treated with a mixture of penicillin and dihydrostreptomycin sulfate given locally and parenterally with satisfactory results. Only one patient reported a recurrence; one patient failed to receive antibiotic therapy because he did not follow instructions.

Since this is a preliminary report, it is hoped that it will serve to stimulate further studies and studies with longer periods of observation, so that a final evaluation of results can be attained.

11. Cruz, A.: Personal communication to the author.

CLINICAL PROBLEMS PERTAINING TO NEUROTOXICITY OF STREPTOMYCIN GROUP OF DRUGS

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THE MOST important complications arising from the therapeutic use of streptomycin and dihydrostreptomycin are severe damage to the vestibular and cochlear mechanisms. Dihydrostreptomycin, which is hydrogenated streptomycin, has no therapeutic advantage over streptomycin. It was developed with the hope that it might not produce the vestibular and cochlear complications which are so frequent with the parent drug.

These antibiotics are most frequently employed by the chest physician, the general surgeon, and the urological surgeon; the otolaryngologist relatively rarely employs these agents in his practice. The otolaryngologist is, however, frequently consulted with regard to the vestibular and cochlear complications but, unfortunately, in many instances after streptomycin or dihydrostreptomycin therapy has been started or has been in progress for two or more weeks. For example, a patient who has received 1 gm. of streptomycin on one day finds that he is unable to hear his watch tick in the left ear the day following and emphatically insists that he never had any impairment in his hearing before streptomycin therapy was started. The otolaryngologist is then faced with the query, Could 1 gm. of streptomycin be responsible for this hearing impairment? No audiometric studies had been done before therapy was started. Likewise, the otolaryngologist is requested to evaluate the vestibular status in a patient who has been receiving streptomycin therapy for two or three weeks. No preliminary vestibular examination had been made. It is almost needless to say that it would be so much simpler and scientifically more accurate for the otolaryngologist to detect the onset of cochlear and vestibular damage produced by these antibiotics if he had a preliminary audiogram and vestibular data for comparison. I do not lose sight of the fact that in some instances the patient's condition does not permit one to make a preliminary audiogram and vestibular examination.

The otolaryngologist is sometimes faced with the problem of the tuberculous pregnant woman and the query, "What effect will streptomycin therapy have on the cochlear and vestibular apparatus of the fetus?" Again, the otolaryngologist is faced with the query, "Would it not be scientifically sound to use streptomycin or dihydrostreptomycin in the treatment of Ménière's disease?" It is the purpose of this paper to present some clinical and animal investigative data and to mention some of the peculiarities of the cochlear and vestibular complications resulting from

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streptomycin and dihydrostreptomycin therapy which will enable the otolaryngologist to answer these queries intelligently whenever they arise.

A report adopted by the Council on Pharmacy and Chemistry of the American Medical Association on 543 tuberculous patients who were given approximately 2 gm. or more of streptomycin daily showed that deafness developed in 1.1% and vestibular symptoms in 96%.[1] The cochlear and vestibular complications are always bilateral, and when the damage is slight or moderate, improvement may subsequently take place. When the damage is severe, the cochlear and vestibular dysfunction is largely permanent. Brown and Hinshaw found that with streptomycin tinnitus usually precedes deafness and that tinnitus followed by deafness practically never occurs with daily doses of less than 3 gm. Deafness is of the perceptive type.[2] I have observed a moderate degree of vestibular dysfunction during therapy in a patient who was given 1.8 gm. of streptomycin daily for 23 days. When the vestibular tests were repeated one week after therapy was discontinued the dysfunction had become severe.[3]

On the other hand, dihydrostreptomycin damages the cochlear mechanism much more frequently than does streptomycin. Of a total of 48 patients treated with dihydrostreptomycin, reported by Carr and associates,[4] Bernard, Paley, and Arnaud,[5] and Shane and Laurie,[6] hearing impairment developed in 18, or 37.5%. In one patient in Shane and Laurie's series deafness came on 24 days after therapy was stopped. In a series of 211 tuberculous patients who were treated with dihydrostreptomycin, Glorig found that a perceptive deafness of 30 db. or more developed in 31%. Twelve patients in his series became deaf one to five months after therapy was discontinued.[7] Dihydrostreptomycin is said to be somewhat less injurious to the vestibular apparatus than is streptomycin. Edison and co-workers[8] and Hobson and associates[9] found that in cats ataxia and the loss of nystagmus on turning came on later and were somewhat less marked than with streptomycin. My associates and I have found no such difference in cats. Ataxia often appeared within 24 to 48 hours, and there was a loss of the eye response to turning at the

1. Streptomycin in Treatment of Infections: Report of 1,000 Cases, National Research Council, Committee on Chemotherapeutics and Other Agents, J. A. M. A. **132**:4 (Sept. 7) 1946; **132**:70 (Sept. 14) 1946.

2. Brown, H. A., and Hinshaw, H. C.: Toxic Reaction of Streptomycin on the Eighth Nerve Apparatus, Proc. Staff Meet., Mayo Clin. **21**:347 (Sept. 4) 1946.

3. Winston, J.: The Vestibular Findings Before, During, and After Vestibular Damage Resulting from Streptomycin Therapy, Arch. Otolaryng. **47**:746 (June) 1948.

4. Carr, D. T.; Brown, H. A.; Hodgson, C. H., and Heilman, F. R.: Neurotoxic Reactions to Dihydrostreptomycin, J. A. M. A. **143**:1223 (Aug. 5) 1950.

5. Bernard, E.; Paley, P. Y., and Arnaud, G.: Sur l'action toxique cochléo-vestibulaire de la dihydrostreptomycine, Bull. et mém. Soc. méd. hôp. Paris **66**:583 (April) 1950.

6. Shane, S. J., and Laurie, J. H.: Neurotoxicity of Dihydrostreptomycin, Canad. M. A. J. **63**:277 (Sept.) 1950.

7. Glorig, A.: The Effect of Dihydrostreptomycin Hydrochloride and Sulfate on the Auditory Mechanism, Ann. Otol. Rhin. & Laryng. **60**:327 (June) 1951.

8. Edison, A. O.; Frost, B. M.; Graessle, O. E.; Hawkins, J. E., Jr.; Kuna, S.; Mushett, C. W.; Silber, R. H., and Solotorovsky, M.: An Experimental Evaluation of Dihydrostreptomycin, Am. Rev. Tuberc. **58**:487 (Nov.) 1948.

9. Hobson, L. B.; Tompsett, R.; Muschenheim, C., and McDermott, W.: A Laboratory and Clinical Investigation of Dihydrostreptomycin, Am. Rev. Tuberc. **58**:501 (Nov.) 1948.

end of 14 days in all cats who had received 1 gm. of streptomycin or dihydrostreptomycin daily.[10] Carr and his colleagues reported vestibular damage in all of 10 patients who received dihydrostreptomycin therapy.[4] The incidence of cochlear and vestibular complications is directly proportional to the size of the daily dose of streptomycin or dihydrostreptomycin and to the number of days that such daily doses are administered. These complications appear more or less abruptly with dihydrostreptomycin and more gradually with streptomycin. The site of the damage to the vestibular and cochlear mechanisms produced by these antibiotics has been in considerable disagreement by various investigators. Glorig and Fowler Jr.,[11] Berg,[12] and Hawkins and Lurie [13] are among those whose clinical or animal investigations lead them to believe that the locale of the damage is in the end-organ only, while Stevenson, Alvord, and Correll,[14] my co-workers and I,[15] Floberg, Hamberger, and Hydén,[16] and Christensen and co-workers [17] are among those who have demonstrated in human and/or animal material that these antibiotics damage the central cochlear and vestibular mechanisms in the brain stem and cerebellum. It is quite possible that these antibiotics may damage both the central and the peripheral parts of the vestibular and cochlear mechanisms. Clinically, the turning, or centrifugal, stimulus repeated at weekly intervals is useful in making a quantitative estimation of the effects of streptomycin and dihydrostreptomycin upon the vestibular function during the course of therapy. Since there is a variation in the duration of nystagmus and vertigo in normal subjects after repetition of the turning tests at weekly intervals, Rubin, Winston, and associates have made nomograms for the nystagmus and vertigo which show when these variations are significant.[18] The caloric stimulus, in which water is used at a fixed number of degrees below or above normal body temperature, is useful only as a qualitative

10. Winston, J.; Lewey, F. H.; Parenteau, A.; Marden, P. A., and Cramer, F. B.: An Experimental Study of the Toxic Effects of Streptomycin on the Vestibular Apparatus of the Cat: Central Nervous System, Ann. Otol. Rhin. & Laryng. **57**:738 (Sept.) 1948; Further Experimental Studies of the Toxic Effects of Streptomycin on the Central Vestibular Apparatus of the Cat, ibid. **58**:988 (Dec.) 1949. Winston, J.; Lewey, F. H.; Parenteau, A.; Spitz, E., and Marden, P. A.: Toxic Effects of Dihydrostreptomycin upon the Central Vestibular Mechanism of the Cat, Ann. Otol. Rhin. & Laryng. **62**:121 (March) 1953.

11. Glorig, A., and Fowler, E. P., Jr.: Tests for Labyrinth Function Following Streptomycin Therapy, Ann. Otol. Rhin. & Laryng. **56**:379 (June) 1947; Tr. Am. Otol. Soc., 1947.

12. Berg, K.: The Toxic Effect of Streptomycin on the Eighth Cranial Nerve: Histological Investigations; A Preliminary Report, Ann. Otol. Rhin. & Laryng. **58**:488 (June) 1949.

13. Hawkins, J. E., Jr., and Lurie, M. H.: The Ototoxicity of Streptomycin, Ann. Otol. Rhin. & Laryng. **61**:789 (Sept.) 1952.

14. Stevenson, L. D.; Alvord, E. C., Jr., and Correll, J. W.: Degeneration and Necrosis in Eighth Cranial Nuclei Caused by Streptomycin, Proc. Soc. Exper. Biol. & Med. **65**:86 (May) 1947.

15. Winston.[3] Footnote 10.

16. Floberg, L. E.; Hamberger, C. A., and Hydén, H.: Inhibition of Nucleic Acid Production in Vestibular Nerve Cells by Streptomycin, Acta oto.-laryng., Supp. 75, p. 36, 1949.

17. Christensen, E.; Hertz, H.; Riskaer, N., and Vra-Jensen, G.: Histological Investigations in Chronic Streptomycin Poisoning in Guinea Pigs, Ann. Otol. Rhin. & Laryng. **60**:343 (June) 1951.

18. Rubin, A.; Winston, J.; Rubin, H. M., and Berwick, L.: The Vestibular Responses to Turning with Nomograms for the Detection of Streptomycin and Other Drug Toxicities and for the Prediction of the Normal Variations of Nystagmus and Vertigo, Ann. Otol. Rhin. & Laryng. **60**:108 (March) 1951.

method of estimating the presence of severe damage to the vestibular mechanism, since the patients to whom these antibiotics are administered are usually afflicted with a febrile illness, such as active tuberculosis, in which the body temperature varies from morning to afternoon and from day to day. The intensity of the caloric stimulus employed will therefore vary with each daily variation of the patient's body temperature. It will also vary from patient to patient.

For example, if the vestibular apparatus are examined at weekly intervals with the Fitzgerald-Hallpike technique,[19] which Glorig and Fowler used in a series of tuberculous patients,[11] in which cool water at 30 C. and warm water at 44 C. are used to stimulate the horizontal semicircular canals, the cool water in a subject with a normal temperature of 37 C. will be exactly 7 degrees below body temperature and the warm water will be exactly 7 degrees above body temperature. With patients afflicted with active tuberculosis, a febrile illness in which the body temperature is elevated, for example to 39 C., the cool water will be 9 degrees below and the warm water only 5 degrees above the patient's body temperature, so that the cool stimulus becomes a stronger stimulus and the warm stimulus an extremely mild one. The intensity of these stimuli may naturally vary from examination to examination in the same patient, just as the patient's temperature may vary from day to day as the vestibular examinations are made. The intensity of these stimuli will also vary from patient to patient. For example, the temperature of one patient may be 39 C. and the temperature of the next patient 38 C. or 40 C. In 1946 I had the opportunity to make vestibular examinations on two tuberculous patients before, during, and after streptomycin therapy.[3] From repeated turning tests at approximately weekly intervals it was found that in one patient there was a gradual reduction in duration to an appreciably greater degree in the after-turning nystagmus than in the after-turning vertigo. In the second patient reverse changes took place; i. e., the after-turning vertigo was reduced in duration to a greater degree than the nystagmus. These results indicated that in the first patient the vestibulo-ocular pathways were being damaged to a greater degree than the vestibulocerebellocerebral pathways, while in the second patient the damage as it was taking place involved the vestibulo-cerebellocerebral pathways to a greater degree than the vestibulo-ocular pathways. With partial damage to the end-organ the after-turning nystagmus and vertigo are equally reduced. This clinical experience led me to believe that streptomycin damages the central portion of the vestibular apparatus. The same pathologic phenomenon can be seen when the galvanic stimulus is used. Glorig and Fowler Jr. reported that when the vestibular apparatus of three patients who had received 2 gm. of streptomycin daily for 121 days had been stimulated with a galvanic stimulus of less than 6 ma. a vertigo response was obtained in two but no nystagmus. The vestibular mechanisms of these three patients gave no response to a massive caloric stimulus of 80 cc. of ice water. Their hearing was normal.[11] These results suggest to me that the streptomycin had inflicted greater damage on the vestibulo-ocular pathways of these two patients than on the vestibulocerebello-cerebral pathways and, again, that streptomycin damages the central vestibular

19. Fitzgerald, G., and Hallpike, C. S.: Studies in Human Vestibular Function: Observations on the Directional Preponderance ("Nystagmusbereitschaft") of Caloric Nystagmus Resulting from Cerebral Lesions, Brain **65**:115 (June) 1942.

mechanism. They also suggest that all the central vestibular nuclei and pathways are not involved to the same degree.[20] This subtotal damage to the central portion of the vestibular mechanism produced by the administration of streptomycin and dihydrostreptomycin is borne out by the experimental work of my co-workers and me with the cat.[10]

In a large series of cats in whom the vestibular eye response to turning was abolished by the administration of 1 gm. of streptomycin or dihydrostreptomycin daily for 14 days, 59.2% of the streptomycin-intoxicated cats and 100% of the dihydrostreptomycin-intoxicated cats showed microscopic evidence of pathology involving the central vestibular mechanism. Of these nearly all showed definite pathologic changes in the vestibular portion of the cerebellum; one-sixth showed pathologic changes in the vestibular nuclei of the brain stem, and one-third had damage to the central cochlear mechanism in the brain stem. In many of the pathologic areas an occasional normal-appearing brain cell could be seen among numerous degenerated cells. It can, therefore, be clearly seen that the damage to the central vestibular and cochlear mechanisms is subtotal, that the vestibular centers in the cerebellum and the brain stem may not be uniformly involved in degree in all subjects, and that the central cochlear mechanism may not always be involved.

In 1946 Molitor and his co-workers reported that repeated doses of streptomycin administered to weanling rats caused retardation of growth and pronounced nervous hyperexcitability, while no such changes were observed in adult rats.[21] These observations suggest that the immature subject may be more susceptible to the neurotoxic effects of streptomycin than the adult.

The reports of Woltz and Wiley[22] and Heilman and co-workers[23] indicate that streptomycin crosses the placenta. As a result of these reports, in collaboration with Rubin and Rutledge I observed three infants for 16 to 19 months postnatally.[24] The mothers, afflicted with active tuberculosis, had received 1 gm. of

20. The galvanic stimulus is capable of stimulating the vestibular nerve directly as well as the end-organ. The caloric and turning, or centrifugal, stimuli cause a movement of the endolymph in the membranous labyrinth and thus stimulate only the vestibular end-organ. In the presence of a nonfunctioning end-organ but a normal vestibular nerve and central vestibular pathways and nuclei, the galvanic stimulus can bypass the end-organ and stimulate the vestibular nerve directly, producing under these conditions the two vestibular phenomena, namely nystagmus and vertigo. The vestibular nerve fibers on entering the brain stem and reaching the vestibular nuclei divide into two separate pathways: (1) a vestibulocerebellocerebral pathway for the conduction of vertigo-producing impulses and (2) a vestibulo-ocular pathway for the transmission of nystagmus-producing impulses. (Jones, I. H., and Fisher, L.: Equilibrium and Vertigo, Philadelphia, J. B. Lippincott Company, 1918.)

21. Molitor, H.; Graessle, O. E.; Kuna, S.; Mushett, C. W., and Silber, R. H.: Some Toxicological and Pharmacological Properties of Streptomycin, J. Pharmacol. & Exper. Therap. **86**:151 (Feb.) 1946.

22. Woltz, J. H. E., and Wiley, M. M.: Transmission of Streptomycin from Maternal Blood to the Fetal Circulation and the Amniotic Fluid, Proc. Soc. Exper. Biol. & Med. **60**:106 (Oct.) 1945.

23. Heilman, D. H.; Heilman, F. R.; Hinshaw, H. C.; Nichols, D. R., and Herrell, W. E.: Streptomycin: Absorption, Diffusion, Excretion and Toxicity, Am. J. M. Sc. **210**:576 (Nov.) 1945.

24. Rubin, A.; Winston, J., and Rutledge, M. L.: Effects of Streptomycin upon the Human Fetus, A. M. A. Am. J. Dis. Child. **82**:14 (July) 1951.

streptomycin daily for three or more weeks just before delivery. The vestibular and cochlear function of the infants and of the mothers before and after delivery was normal. In two clinical reports by Watson and Stow [25] and Kistner [26] a total of seven infants whose mothers received streptomycin during pregnancy were similarly observed for 2 to 11 months after birth and were said to be normal.

An experience with a total of 10 infants may fail to prove that the fetus is safe from the neurotoxic effects of streptomycin when the mother receives the drug. It does, however, strongly suggest that such may be the case.

Since streptomycin nearly always damages the vestibular apparatus, its use in the treatment of Ménière's disease has been suggested by Floberg, Hamberger, and Hydén [16] and more recently by Hanson.[27] Dihydrostreptomycin should never be used for this purpose because it so frequently damages the cochlear apparatus. In streptomycin the otologist feels that he has a drug which is capable of accomplishing the same end as the surgical treatments without the risks of surgery. In the surgical treatment of Ménière's disease the complete functional destruction of the end-organ involved is the goal. It must be borne in mind that the same results will be accomplished if the central vestibular mechanism alone or the central and the peripheral portions of the vestibular mechanisms are both completely destroyed. I have previously pointed out that streptomycin and dihydrostreptomycin destroy the central vestibular mechanism subtotally.

Mollison had attempted to destroy the vestibular end-organ by injecting alcohol into the membranous horizontal semicircular canal.[28] Day more recently has been accomplishing the same result by substituting the electrocoagulating current for alcohol. Dandy had sectioned the vestibular branch of the eighth nerve on the same side if one labyrinth was involved and bilaterally when both labyrinths were involved.[29]

There are some failures with the methods of Mollison and Day. First, the function of the labyrinth operated upon is not always completely destroyed. With slight residual function, however, the ensuing attacks of vertigo will be mild. Secondly, in some cases of Ménière's disease both labyrinths are involved. These surgical procedures nearly always severely damage the cochlear function and for this reason cannot be performed bilaterally. Hanson in his report on the use of streptomycin in the treatment of five patients suffering from Ménière's disease states that each of the patients had attacks of vertigo many months after completion of therapy but that these attacks were milder.[27] Lurie had, and I believe very wisely so, suggesed that this drug should not be used in the treatment of Ménière's disease because of the damage to the central nervous system which it produces.[30]

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25. Watson, E. H., and Stow, R. M.: Streptomycin Therapy: Effects on Fetus, J. A. M. A. **137**:1599 (Aug. 28) 1948.

26. Kistner, R. W.: The Use of Streptomycin During Pregnancy, Am. J. Obst. & Gynec. **60**:422 (Aug.) 1950.

27. Hanson, H. V.: The Treatment of Endolymphatic Hydrops (Ménière's Disease) with Streptomycin, Ann. Otol. Rhin. & Laryng. **60**:676 (Sept.) 1951.

28. Mollison, W. M.: The Treatment of Vertigo by Destruction of the Labyrinth with Absolute Alcohol, Guy's Hosp. Rep. **80**:470 (Oct.) 1930.

29. Dandy, W. E.: Ménière's Disease: Symptoms, Objective Findings and Treatment in 42 Cases, Arch. Otolaryng. **20**:1 (July) 1934; Surgical Treatment of Ménière's Disease,

30. Lurie, M. H., in discussion on Glorig, A., and Fowler, E. P., Jr.: Tests for Labyrinth Function Following Streptomycin Therapy, Tr. Am. Otol. Soc. p. 86, 1947.

SUMMARY AND CONCLUSIONS

The streptomycin group of drugs frequently damage the cochlear and vestibular mechanisms centrally and possibly the peripheral end-organ also. This damage is always bilateral, and the deafness is of the perceptive type. Streptomycin frequently damages the vestibular apparatus and rarely damages the cochlear apparatus, while dihydrostreptomycin frequently causes deafness as well as severe damage to the vestibular apparatus. The incidence of these complications is directly proportional to the size of the daily dose of the drug and to the number of days it is administered. With streptomycin deafness is usually preceded by tinnitus as a warning symptom. It practically never occurs with daily doses of less than 3 gm. Deafness and vestibular damage are more abrupt with dihydrostreptomycin, and these complications may become more severe or be entirely delayed until some time after therapy has been discontinued. If therapy is discontinued when the complications become manifest and are slight or moderate, the hearing impairment and vestibular dysfunction may ultimately improve. The detection of early vestibular and cochlear complications can best be effected if preliminary audiometry and vestibular turning tests are carried out, from time to time during therapy and again within six months after therapy has been discontinued. Many clinicians fail to see the importance of, or at least fail to request, audiometry and vestibular examinations before starting streptomycin or dihydrostreptomycin therapy. While streptomycin appears to have a much more frequent neurotoxic effect upon immature rats than adult rats and while it has been shown to cross the placenta, no neurotoxic effect upon the fetus has thus far been noted. It would seem, therefore, that pregnancy does not contraindicate the use of streptomycin. In the treatment of Ménière's disease streptomycin does not eliminate the recurrent spells of vertigo. The paroxysms of vertigo are rendered much less severe. Because it attacks the central vestibular mechanism and possibly other brain structures as well, it should not, in my opinion, be used for this purpose.

EFFECT OF PROLONGED EXPOSURE TO INTENSE NOISE ON HEARING ACUITY

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INTRODUCTION

HEARING impairment resulting from exposure to industrial noise was appreciated as far back as 1831 by Fosbroke.[1] Since then this condition has been variously termed occupational deafness, industrial deafness, or acoustic trauma. It has been incontestably demonstrated in certain employees engaged as boilermakers, weavers, and chippers and in persons exposed to small-arms gunfire.

Even prior to the development of the audiometer many otologists recognized the general characteristics of occupational deafness, particularly the loss in high-tone acuity as demonstrated by the subject's inability to hear the whispered voice. Among these early investigators were Gottstein and Kayser,[2] Lake,[3] Wittmaack,[4] Röpke,[5] Barr,[6] and Laurens.[7] With the advent of the audiometer and the tremendous increase in industrial noise, considerable effort has been made to determine the intensity and type of impairing noise and criteria significant in the production of acoustic trauma.

During the past decade the objective of numerous investigations has been to determine a maximum safe intensity level; i. e., a physical sound level below which noise could be considered harmless to the hearing. There is general acceptance that very intense noise can produce permanent hearing impairment, but there is considerable disagreement as to the degree of intensity required to produce such impairment, as well as to the cumulative effect of continued exposure to noise of moderate intensity, which, for the purpose of this report, shall be considered noise with an over-all sound level ranging between 90 and about 120 db. re 0.0002 dyne sq. cm.

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1. Fosbroke, J.: Practical Observations on the Pathology and Treatment of Deafness, Journal-Lancet **19**:645-648, 1831; cited by Kryter, K. D.: J. Speech & Hearing Disorders, Monogr. Supp. 1, 1950, p. 37.

2. Gottstein, J., and Kayser, R.: On Hearing Defects in Locksmiths and Blacksmiths, Breslau. aerztl. Ztschr. **3**:205-207, 1881.

3. Lake, R.: Boiler-Makers' Deafness, J. Laryng. & Otol. **38**:323-324, 1923.

4. Wittmaack, K.: On Injuries to the Ear Caused by the Action of Noise: An Experimental Study, Ztschr. Ohrenh. **54**:37-80, 1907.

5. Röpke, F.: Monograph: Die Berufskrankheiten des Ohres und der oberen Luft Wege, Wiesbaden, J. F. Bergmann, 1902.

6. Barr, T.: Manual of Diseases of the Ear, Glasgow, Maclehose & Sons, 1896, p. 37.

7. Laurens, G.: Oto-Rhino-Laryngology, Baltimore, William Wood & Company, 1919, pp. 108 and 316.

Bunch [8] in 1942 stated that noise above an 80 to 90 db. level was injurious. He concluded that the boundary line between noises which cause hearing losses and those which do not lies between 80 and 90 db. Schweisheimer,[9] studying the effects of noise in the textile industry, considered that the hazard level falls between 80 and 90 db. Goldner [10] stated that a consensus seemed to indicate that the minimum safe level is in the neighborhood of 80 db. McCord and Goodell [11] in 1943 suggested that a level of 80 to 85 db. would cause some defects of hearing in the high-frequency zones after a period of years. The same authors in conjunction with McCoy [12] in 1944 concurred that levels over 100 db. are a first concern. Rosenblith [13] in 1942 admitted difficulty in specifying a danger level but believed as little as 75 or 85 db., if sufficiently prolonged, would suffice to bring about premature aging of the ear. In an extensive study, MacLaren and Chaney [14] stated, "The dividing line between hazardous and nonhazardous noise lies between 70 and 100 db." The quantitative and qualitative uncertainty existing in this field is revealed by Kryter,[15] who concluded, after what is probably the most astute and comprehensive review of the literature yet made on the deafening effects of noise, that "the 'guess' that tones 85 db. above .0002 dynes/cm. may cause some deafness, either temporary or permanent, applies only for long periods of exposure, applied intermittently over months or years."

In 1945 Davis [16] stated, "There is no rigid proof of permanent impairment of hearing from noise of less than 115 to 120 db.—concern about noise levels below 100 db. is unwarranted." Subsequently, in 1947, he [17] reported that "Noise of less than 100 db. may reasonably be considered quite safe except perhaps for a few unusually susceptible individuals." My investigations have led me to concur strongly in the latter conclusions.

The conflicting experimental conclusions are, in my opinion, the result of failure to take into account, during the many investigations to establish maximum safe intensity levels, the additional criteria that need to be considered before conclusions can be drawn that are valid and reliable.

It is of the greatest importance, for example, that there be accurate calibration of instruments used for sound measurement, such as audiometers, sound-level meters and sound analyzers. It is essential that all equipment be of the proper type and accurately calibrated at the time of use. The limitations of each piece of equipment must be fully understood by the person using it. Such understanding is

8. Bunch, C. C.: Conservation of Hearing in Industry, J. A. M. A. **118**:588-593, 1942.

9. Schweisheimer, W.: Effects of Noise in the Textile Industry, Rayon Text. Mon. **26**:593, 1945.

10. Goldner, A.: Occupational Deafness: With Special Reference to Chronic Occupational Deafness, Arch. Otolaryng. **42**:407-411, 1945.

11. McCord, C. P., and Goodell, J. D.: Abatement of Noise, J. A. M. A. **123**:476-480, 1943.

12. McCoy, D. A.: The Industrial Noise Hazard, Arch. Otolaryng. **39**:327-330, 1944.

13. Rosenblith, W. A.: Industrial Noises and Industrial Deafness, J. Acoust. Soc. Am. **13**:220-225, 1942.

14. MacLaren, W. R., and Chaney, A. L.: An Evaluation of Some Factors in the Development of Occupational Deafness, Indust. Med. **16**:109-115, 1947.

15. Kryter, K. D.: Speech & Hearing Disorders, Monogr. Supp. 1, 1950, p. 37.

16. Davis, H.: Protection of Workers Against Noise, J. Indust. Hyg. & Toxicol. **27**:56-57, 1945.

17. Hearing and Deafness, edited by H. Davis, New York, Rinehart & Co., Inc., 1947, p. 95.

especially important when microphones are used under different conditions of temperature and pressure. Beranek [18] emphasizes the importance of this consideration when he describes "The complex character of the science of sound."

Too often the investigator, having an excellent appreciation of the electronic and physical aspects of sound measurement, takes for granted the apparent simplicity of taking and interpreting audiograms. More often the situation is reversed, and the competent otologist, assuming that he has merely to read a meter, draws important conclusions which may be based on inaccurate sound measurements.

Undoubtedly the greatest cause for the lack of valid experimental conclusions as to the effect of industrial noise upon hearing acuity is the failure of the investigator to appreciate the scientific limitations of the audiogram. In my opinion a single audiogram is inadequate to establish a subject's threshold of hearing. It is inadvisable to draw conclusions concerning the change in hearing acuity on the basis of a single audiogram taken prior to exposure and a single audiogram taken after exposure to industrial noise. This statement is substantiated by the significant variation in threshold often obtained for the same subject by the same tester under standard conditions when audiograms are repeated several minutes apart. A single audiogram is particularly unreliable when there are a large number of subjects to be tested one after another. At least two and often more separate independent audiograms which are consistent with each other are essential to establish a threshold of hearing.

The attitude and interest of the technician are also important factors in determining thresholds. Whenever possible, it is advisable for the competent investigator to perform his own audiometry and not rely on a technician except when the latter is responsible for and intensely interested in the investigation. The taking of routine audiograms one after another for numerous subjects is too readily conducive to neglect of some of the vital aspects of good audiometry.

A number of reports in the literature represent much labor and thousands of hearing tests, but too frequently the tests were not made under standard conditions and were not reliable determinations of the subject's threshold both before and after exposure. The ambient-noise level of the test room should not interfere with the performance of audiometry and should be standard for the entire investigation. The ambient-noise level also needs to be accurately measured and evaluated.

In a long-range study of the deafening effects of noise, it is important to consider the time of day when the testing is done. If, for example, a subject is employed as a chipper, his hearing threshold will probably be considerably different in the morning prior to work from what it will be after six hours of exposure. It is essential in a prolonged study to consider previous exposure and compare hearing thresholds taken at the same relative time of day, so that the initial determination is not made in the morning and the final hearing evaluation in the evening after exposure. In order to determine accurately the existence of permanent acoustic trauma it is necessary that hearing thresholds be carefully stabilized.

Until very recently almost all interested investigators have attempted to evaluate the deafening effect of an industrial noise on the basis of over-all measures of

18. Beranek, L. L.: Sound and Its Measurement, A. M. A. Arch. Indust. Hyg. **5**:97-108, 1952.

intensities. The inadequacy of the approach has been demonstrated by such investigators as Kryter [15] and Williams,[19] who have shown that the spectrum of a noise must be carefully analyzed before its deafening effect can be determined. Neglecting to consider noise analysis accounts for many of the conflicting opinions regarding maximum safe intensity level. Canfield [20] has stated, "When we ask how much pressure will the human ear tolerate, we must define our question by further analyzing the types of sound to which the human ear is exposed. The elements of frequency, duration and intensity must all be considered."

A careful consideration of frequency analysis is particularly vital when one is dealing with noise in which the energy is mostly in the low or high frequencies, because, as Kryter [21] points out, "Noise-level meters as commonly used add together the pressures present in a noise throughout the range of sound frequencies . . . . " Perlman [22] has experimentally demonstrated that sounds of low frequency produce less acoustic trauma than those of high frequency. Grove [23] and many other authors have clinically shown that noise in which the sound energy is predominantly in the high frequencies is more harmful to hearing than noise in which the energy is predominantly in the lower frequencies. Beranek [24] has established a damage risk curve indicating that sound pressure above this curve could result in permanent deafness if a person were subjected to the pressure levels for a prolonged period. The validity of this curve will be determined by the tests of time and usage. The importance of correlating sound-level measurements with spectral analysis of the noise is obvious. For both these determinations it is essential to make the readings in relation to the position of the ear at the time of exposure.

Besides these essential criteria in studying the deafening effect of noise upon hearing, one must consider the time relations of a sound stimulus, such as its period of continuity, fluctuation and rate of build-up and release of sound energy. In addition, there are the disturbing questions of the individual's susceptibility and adaptability to the noise, about which at present very little is understood.

The cumulative character of acoustic trauma has also introduced considerable conflicting opinions, most of which are admittedly not based on rigid experimental data. Machle,[25] for example, believes that with repeated equal exposures acoustic damage is cumulative, apparently largely because recovery from initial traumatic response is incomplete in the 24-hour intervals between exposure. Emphasizing temporary hearing loss, Davis and associates[26] state, "Certainly, a very considerable temporary deafness may be incurred repeatedly without demonstrable cumulative effect." This opinion is substantiated by the outstanding investigation of Davis

19. Williams, C. R.: Industrial Plant Environmental Study, A. M. A. Arch. Indust. Hyg. **5**:109-112, 1952.

20. Canfield, N.: Noise in Industry, speech presented at Institute of Occupational Medicine, Yale University, March 30, 1948.

21. Kryter, K. D.: Noise Safety Criteria, A. M. A. Arch. Indust. Hyg. **5**:117-120, 1952.

22. Perlman, H. B.: Acoustic Trauma in Man: Clinical and Experimental Studies, Arch. Otolaryng. **34**:429-452, 1941.

23. Grove, W. E.: Will Noise Damage One's Hearing? J. A. M. A. **140**:674-677, 1949.

24. Beranek, L.: Personal communication to the author.

25. Machle, W.: Effects of Gun Blasts on Hearing, Arch. of Otolaryng. **42**:164-168, 1945.

26. Davis, H.; Parrack, H. O., and Eldredge, D. H.: Hazards of Intense Sound and Ultrasound, Ann. Otol. Rhin. & Laryng. **58**:732-738, 1949.

and his colleagues[27] in 1943. Yet Guild [28] in 1950 concluded that the bad effect of noise on hearing ability is cumulative. He further believes that "Continued frequent exposures, however, to noises that at first cause transient impairments for hours or days, eventually cause permanent impairments."

The relation of temporary hearing impairment to permanent damage is still not clearly understood. The experience reported by Eldredge and Parrack [29] is not uncommon. They note that "During the past nine years one man working in this field (jet-engine noise) has sustained many severe temporary hearing losses with no audiometric indication of permanent damage to his ears." However, it is not my intention in the present report to delve into this problem except to interject the statement that several experiments tangent to the present investigation and soon to be published provide pertinent experimental data on this relationship.

The distinction between high-tone loss produced by exposure to intense noise and that resulting from presbycusis [30] is also not clear. MacLaren and Chaney [14] believe that the types of hearing loss can be distinguished by the frequencies affected. These authors state, "In contrast, the loss associated with exposure to noise appears to involve the frequencies from 3000 to 5000 cycles, while frequencies above and below are heard relatively well." There is considerable difference of opinion concerning this concept.

The purpose of this investigation was to study the effect of continuous noise of measured duration and frequency characteristics, with intensity fluctuations in the range between 90 and 120 db., upon hearing acuity after prolonged exposure.

EXPERIMENTAL DESIGN

For the past five years semiannual audiographic and regular otological examinations have been done on 272 employees engaged in testing jet engines at a large industrial plant. Whenever possible, preplacement and preemployment audiograms were taken along with a careful history of previous exposure to acoustic trauma, particularly gunfire.

Audiography was done by me in a standard manner. The tone at 1024 cycles was tested first and followed by every higher octave up to and including 8192. The two lower octaves were then tested, after which the tone at 1024 cycles was retested. The descending method of testing was used, starting with an audible tone.

In obtaining thresholds for 2048 and above at least two and generally three independent determinations were made during the taking of each audiogram. If a subject demonstrated a significant hearing loss, the test was generally repeated by another tester on the same audiometer with the same technique. If an audiogram was inconsistent with previous tests, it was repeated until a stable consistent threshold was established. Masking was not used except in unusual cases of unilateral deafness.

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27. Davis, H. E., and others: Final Report on Temporary Deafness Following Exposure to Loud Tones and Noise, Acta oto-laryng., Supp. 88, 1950.

28. Guild, S. R.: Industrial Noise and Deafness, J. Insur. Med. **5**:32-34, 1950.

29. Eldredge, D. H., Jr., and Parrack, H. O.: Sound Problems in the Air Force, U. S. Armed Forces M. J. **1**:449-461, 1950.

30. House, H. P.: Clinical Aspects of the Problems of Noise in Industry, A. M. A. Arch. Indust. Hyg. **5**:124-128, 1952.

Accurate, stabilized hearing thresholds for all subjects were obtained prior to and after prolonged exposure to a continuous type of occupational noise produced by jet engines. Thresholds for many of the same subjects were also obtained after prolonged periods of nonexposure to the sound.

A normal audiogram is considered one in which there is no loss greater than 10 db. in any octave frequency between 256 and 8192, inclusive. For the purposes of this study the first two octaves were not considered important and were therefore eliminated from evaluation in the results.

A change in threshold for any frequency was considered significant when there was a persistent change of at least 10 db., except for a normal audiogram in which the threshold is 0 or 5 db., in which case a significant change must be at least 15 db.

Only audiograms taken more than 20 minutes after exposure to jet noise were evaluated, in order to obtain stabilized hearing thresholds as little influenced by auditory fatigue as possible.[31]

Determinations were made of the duration of each subject's daily exposure to the sound and the duration of his cumulative exposure throughout the year. Measurements were made of the intensity and frequency characteristics of the noise to which each subject was exposed during various hours of the day over a period of years.

During the entire study all hearing tests were performed in the same room with the ambient-noise level constantly between 50 and 55 db. Retesting more than 50 subjects in a sound-treated room with the ambient-noise level below 30 db. established that the specific type of ambient-noise level of 50 to 55 db. did not significantly affect the threshold values in the octave frequencies between 2000 and 8000 cycles.

Diagnosis for all the abnormal ears included in the results was inner-ear deafness.

It is expected that at least 10 years of continuing study will be required before final data can be analyzed and fully evaluated. Comprehensive noise analyses and hearing evaluations will continue to be performed for subjects during their occupational exposure. Those subjects who show significant hearing losses, particularly of the insidiously progressive type, will be studied during prolonged periods of nonexposure to noise to determine whether the hearing losses are still progressing. The periods of nonexposure are now varying from two weeks to two years. It is anticipated that those subjects whose hearing losses are produced by acoustic trauma will show no further hearing loss or even a slight improvement, whereas the subjects with presbycusis will continue to show progressive loss over several years.

This method would seem to be warranted on the basis of data now available but as yet insufficient to justify conclusions in this preliminary report.

APPARATUS

A single Western Electric Model 6BP audiometer was used throughout the investigation. The calibration of the audiometer was routinely checked on at least 20 normal ears on the days hearing tests were performed for this experiment. The high percentage of normal audiograms in the data partially attests the calibration of the instrument. Data gathered more than five years earlier with another type of audiometer, which did not meet the specifications of calibration, are generally not included in the final results except to corroborate certain normal thresholds.

31. Collateral study incident to the present investigation on 40 subjects (80 ears) revealed no significant change in hearing thresholds obtained 15 minutes after 5 hours of exposure to routine test cell noise described under the heading "Procedure" for Group B. This collateral study did not include any subjects who were exposed to more than 114 db. of noise.

The sound-level readings were made with a calibrated General Radio Sound Level Meter and rechecked with a calibrated Scott Sound Level Meter. The determinations were made with the "C" weighting on the meter, that is, an equal response over the entire frequency range. The spectral analyses were obtained from a General Radio Sound Level Meter coupled to a Panoramic Sonic Analyzer AP-1. The analyses were done with the cooperation of an acoustic engineer employed at the industrial plant and in part confirmed with similar equipment through the cooperation of the Aero-Medical Experimental Laboratory, United States Naval Base, Philadelphia.

PROCEDURE

Sound-level readings were made at frequent intervals in the test-cell areas and were found to vary with the type, speed, and number of engines running and according to whether or not the after-burner was in use. Analysis of the sound levels and correlation with the duration of each subject's daily exposure revealed several natural groups into which almost all the subjects could be placed.

The subjects classified in Group A experienced exposure to the loudest noise in their daily work. The minimum criteria for each group were as follows:

Total Daily Exposure for Each Subject

Group A	
2 hr. daily	104 to 110 db. (yard)
1 hr. daily	91 db.
1 hr. daily	116 db.
5 min. daily	114 db. each day
5 additional hr. per mo. at	116 db.
15 to 45 min. 3 days a week at	120 to 124 db.
Group B	
2 hr. daily	104 to 110 db. (yard)
1 hr. daily	100 db.
½ hr. daily	96 db.
5 min. daily	114 db.
Group C	
104 to 110 db. for about 2 hr. daily	
Group N	
A small number of subjects who had little or no exposure to test-cell noise; N signifies "None"	

All the subjects in Groups A and B were exposed to the minimum criteria at least 5 hours daily, 5 days a week, for about 49 weeks a year. Occasionally one of the engines had to be repaired or changed, which resulted in periods of comparatively little noise. On many occasions, however, the subjects worked seven days a week at much louder noise levels than those specified.

In order to get as complete a description of the jet noise as possible, numerous spectral analyses were obtained in most of the locations where intensity levels were recorded. From these it was hoped that it would be possible to correlate exposure time, intensity levels, and frequency patterns with the hearing loss, if any, produced in each subject.

Figures 1 to 8 are photographs of some of the spectra obtained. Figures 1 to 5, inclusive, were obtained in the yard, where most of the subjects spent at least two hours daily. Although various types of engines have been tested at this industrial

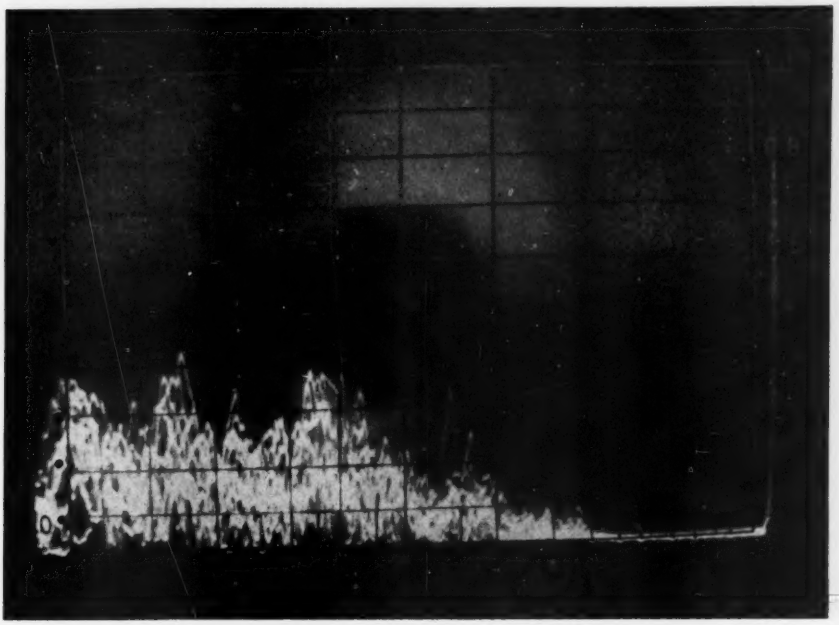

Fig. 1.—Microphone in front of No. 10 cell; engine in Cell 2; over-all sound level 90 db.; zero reference on logarithm scale 90 db.

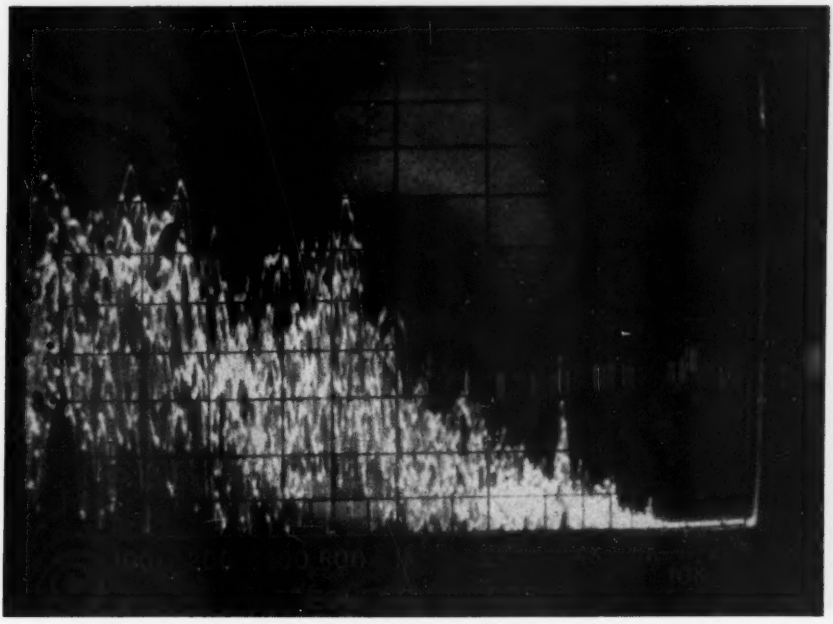

Fig. 2.—Microphone in front of No. 10 cell; engine in Cell 2; over-all sound level 99 db.; zero reference on logarithm scale 90 db.

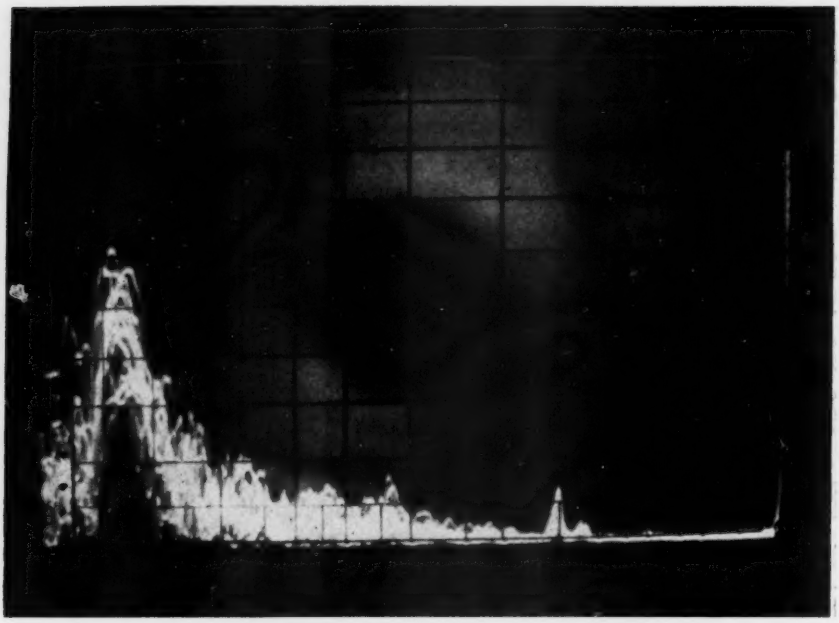

Fig. 3.—Microphone in front of No. 10 cell; engines in Cells 3 and 4; over-all sound level 100.5 db.; zero reference on logarithm scale 100 db.

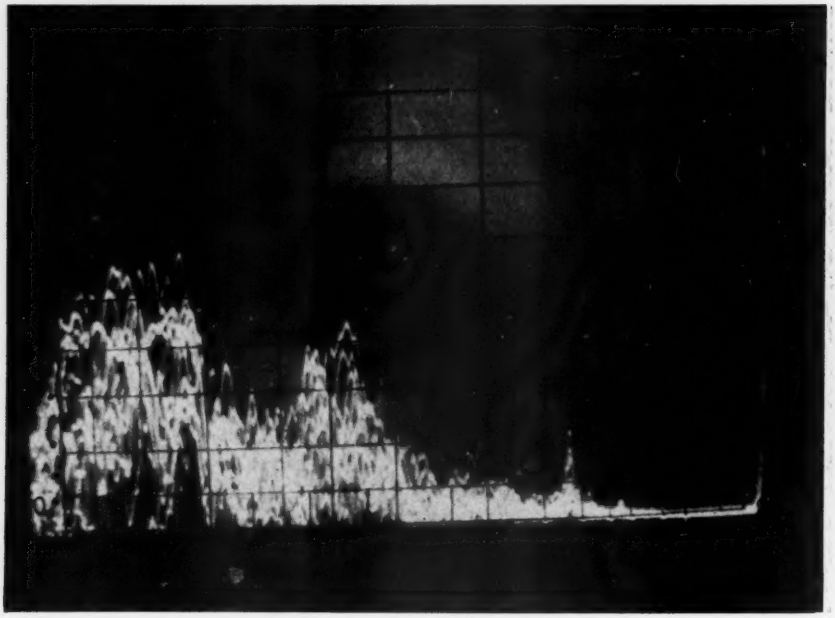

Fig. 4.—Microphone in front of No. 10 cell; engines in Cells 2 and 4; over-all sound level 102 db.; zero reference on logarithm scale 100 db.

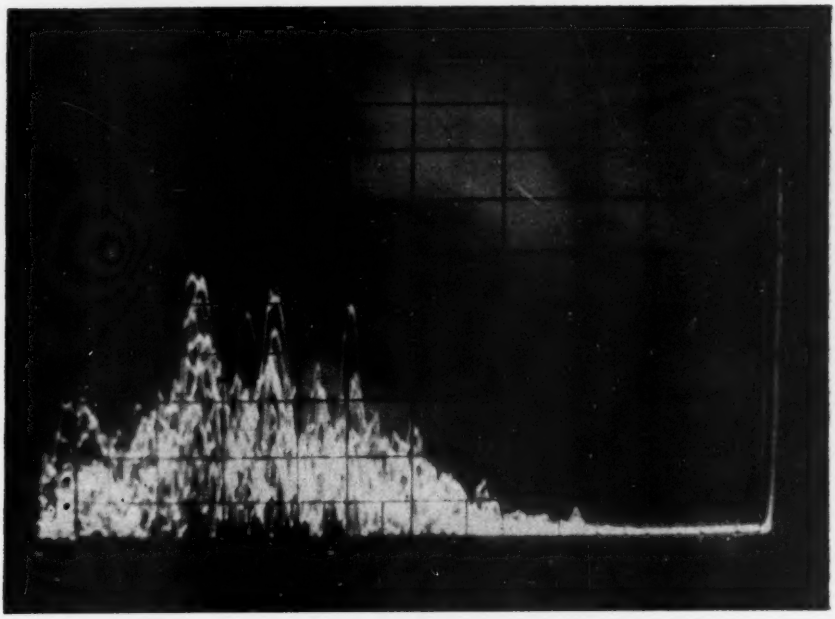

Fig. 5.—Microphone in front of No. 10 cell; engines in Cells 11, 2, and 6; over-all sound level 110 db.; zero reference on logarithm scale 110 db.

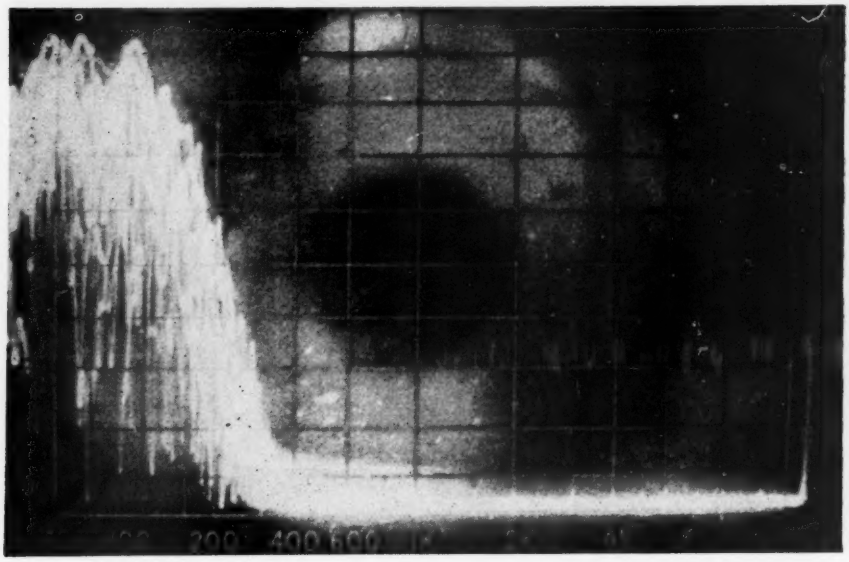

Fig. 6.—Microphone on 45 degree line 15 ft. (391 cm.) from exhaust muffler; over-all sound level 121 db.; zero reference on logarithm scale 102 db.

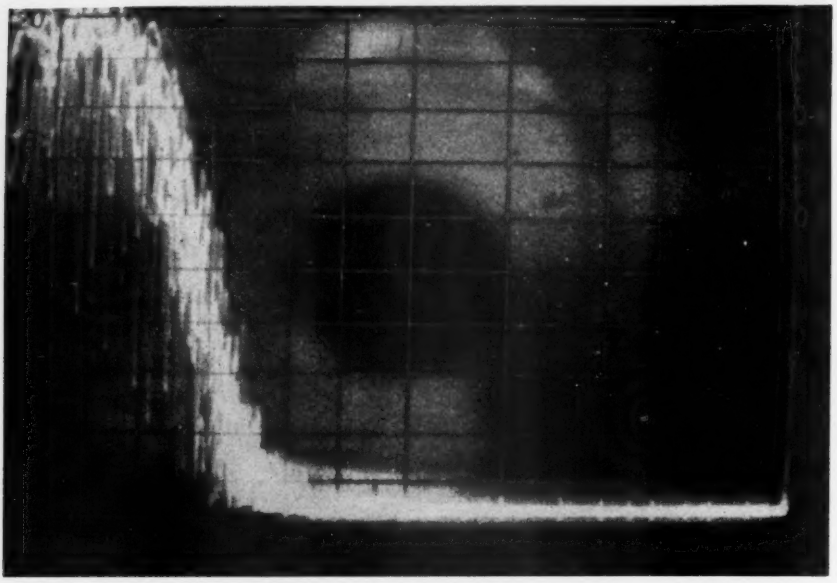

Fig. 7.—Microphone on 45 degree line 15 ft. from exhaust muffler; over-all sound level 110 db.; zero reference on logarithm scale 82 db.

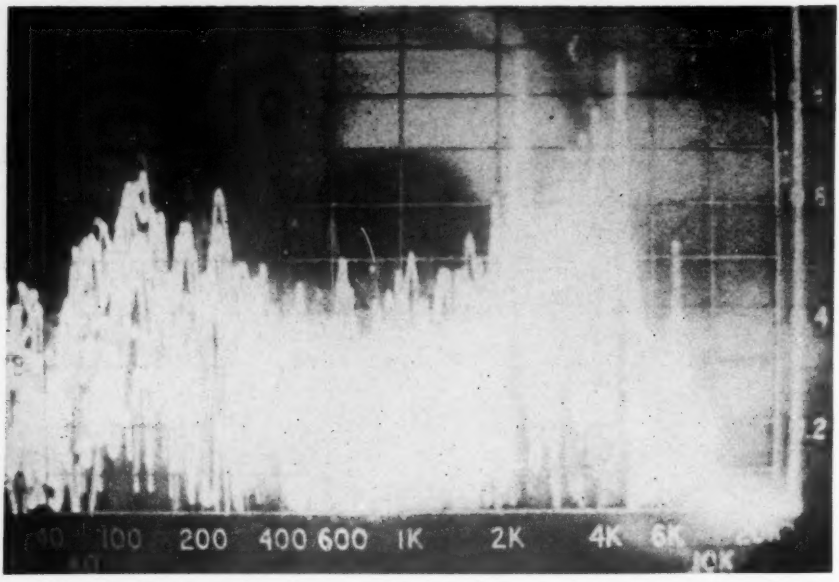

Fig. 8.—Microphone on center line of engine between engine and cell doors; over-all sound level 136 db.; zero reference on logarithm scale 114 db.

plant during the past five years, for the purpose of this study only the findings for two types used continuously during the past two years are presented.

Examination of the spectra shows a basically similar frequency pattern within certain limits. As long as the intensity of the noise remained below about 126 db., there was comparatively little sound energy produced in the frequencies above 1000 cycles. In most instances, the greatest sound energy was concentrated in the frequency range below 600 cycles. It therefore seems proper to suppose for the purpose of this experiment that one was investigating the potentially hazardous effect of exposure to moderately intense noise of low frequency.

TABLE 1.—*Age Distribution of Subjects at Time of Initial Audiograms*

Age, Yr.	N*	A†
20-30	37	42
30-40	21	32
40-50	1	19
50-60	0	3
Total	59	95

* Subjects with normal hearing thresholds at start of investigation.
† Subjects with abnormal hearing thresholds at start of investigation.

TABLE 2.—*Duration of Exposure*

Years Exposed	N*	A†
5	10	35
4	3	7
3	8	7
2	13	13
1	12	19
½	13	14
Total	59	95

* Subjects with normal hearing thresholds at start of investigation.
† Subjects with abnormal hearing thresholds at start of investigation.

RESULTS AND COMMENT

In order to adhere to the most rigid criteria and indices possible, 118 of the 272 subjects were excluded from the present evaluation because of (1) evidence of conductive hearing impairment, (2) use of any sort of ear protector during more than 10% of the daily exposure, and (3) lack of complete data. The remaining 154 subjects were used for the study, and summaries of the data obtained are shown in Tables 1 to 9. When these 154 subjects were first tested, 59 were found to have normal initial hearing thresholds, while there were 95 whose hearing thresholds were abnormal.

Table 1 shows that almost all subjects in this study were under 50 and the great majority under 40 years of age.

An evaluation of the available data reveals certain trends which may well persist throughout the study. Of the 59 subjects whose hearing thresholds were normal at the start of the investigation, 50 showed no change in hearing at any frequency and 9 (15%) showed some audiometric change. Details, summaries, and correlations on these subjects are presented in Tables 3, 4, 7, and 9.

TABLE 3.—*Data on Subjects with Initially Normal Hearing Thresholds: Correlation of Duration of Exposure, Gunfire Exposure, Intensity of Exposure, and Change in Auditory Threshold*

Years Exposed	No. of Subjects	Gunfire	Intensity Group	Threshold Change
5	2	+	A	−
	2	−	B	−
	4	−	B	−
	1	−	C	+
	1	−	N	−
4	1	−	C	−
	1	+	N	−
	1	−	N	−
3	1	−	A	−
	1	+	B	−
	2	+	C	−
	3	−	C	−
	1	−	N	−
2	1	+	A	+
	1	−	A	−
	2	+	B	+
	2	+	B	−
	1	+	C	+
	4	−	C	−
	1	+	C	−
1	4	−	A	−
	1	+	A	+
	1	+	B	−
	2	−	B	−
½	4	+	C	−
	2	+	B	+
	1	+	B	−
	2	−	B	−
	6	+	C	−
	2	−	C	−
	1	+	C	+
Total	59		9 Changed 50 Unchanged	

TABLE 4.—*Threshold Changes Among Nine Subjects of Table 3*

No.	Exposure Group	Yr.	Db. Loss*	Frequency	Ear	Yr. Detected†	Age
1	C	5	5	8	L	5	41
2	A	2	5	4	L	2	42
3	B	2	5	4	L	1	22
4	B	2	10 5	4 8	L	1	37
5	C	2	5	4	R	2	30
6	A	2	5	4	R	2	37
7	B	1	10	4	L	1	39
8	B	1	5	8	L	1	27
9	C	1	5	8	L	1	25

* Decibels lost in excess of expected variation in normal threshold.
† Year in which the significant hearing loss was first detected.

Of the 50 subjects who showed no loss, 20 were exposed for three or more years and 19 were in exposure Groups A and B. Two of the subjects in Group A were among seven who had considerably more than the minimum exposure.

TABLE 5.—*Data on Subjects with Initially Abnormal Hearing Thresholds: Correlation of Duration of Exposure, Intensity of Exposure and Change in Auditory Threshold*

Years Exposed	No. of Men	Gunfire	Exposure	Change
5	5	+	A	−
	2	−	A	−
	1	−	A	+
	2	+	B	+
	6	−	B	−
	8	−	B	+
	1	+	C	−
	4	−	C	+
	4	−	C	−
	1	+	C	+
	1	+	N	+
4	1	−	A	+
	1	+	B	−
	1	+	B	+
	1	−	B	−
	1	+	C	−
	2	+	C	+
3	1	+	A	+
	2	+	B	+
	1	+	B	−
	2	+	C	−
	1	−	C	−
2	1	+	A	−
	1	−	A	+
	1	+	B	−
	3	+	B	+
	2	+	C	−
	2	+	C	+
	2	−	C	+
	1	−	N	−
1	1	+	A	−
	4	+	A	+
	3	+	B	−
	1	+	B	+
	1	−	B	−
	1	−	B	+
	3	+	C	−
	3	+	C	+
	1	−	C	+
	1	−	N	+
½	1	+	A	−
	2	−	A	−
	3	+	B	+
	8	+	C	−
Total	95		46 Changed 49 Unchanged	

Of the nine subjects who showed some audiometric change, one was exposed for five years and the others for two years or less. Two were in Exposure Group A, four in Group B and three in Group C. Six of the nine subjects were over 30 years of age. All of the nine with the exception of Subject 1, who was 41, had previously been exposed to gunfire. It is interesting to note from Table 4 that changes in hearing acuity were first noticed during the first year or early in the second year in most of the nine subjects.

TABLE 6.—*Threshold Changes Among Forty-Six Subjects of Table 5*

No.	Exp.	Dur.	Amt.*	Fr.	Ear	Yr.†	Age	No.	Exp.	Dur.	Amt.*	Fr.	Ear	Yr.†	Age
1	A	5	10 5	4 4	R L	5	34	24	B	3	15	4	L	2	32
2	B	5	25 10	4 8	R L	4	27	25	A	2	20	4	L	1	29
3	B	5	30	4	L	4	46	26	B	2	15 20	4 4	R L	3	22
4	B	5	5	8	R	4	27	27	B	2	5	4	R	1	30
5	B	5	30	8	R	3	40	28	B	2	15 20 10 30	4 8 4 8	L R	1	22
6	B	5	15 5	4 8	R-L L	5	52	29	C	2	10 15	4 4	L R	2	29
7	B	5	10	4	L	4	44	30	C	2	10	4	L	1	25
8	B	5	20	4	L	3	50	31	C	2	10 25	4 4	R L	1	30
9	B	5	5	4	L	5	36	32	C	2	10 15	4 8	L	1	25
10	B	5	5	4	R-L	5	40	33	A	1	15	2	L	1	25
11	B	5	10	4	R-L	5	44	34	A	1	15	4	L	1	36
12	C	5	25 10 15	8 4 8	R L L	5	40	35	A	1	10 15	4 8	L L	2	36
13	C	5	10	8	L	4	33	36	A	1	20	8	L	1	22
14	C	5	5	4	L	5	44	37	B	1	5	4	L	1	37
15	C	5	10	4	L	4	44	38	B	1	10 20	4 8	R	1	28
16	C	5	10	4	L	5	32	39	C	1	20	8 8	L L	1	25
17	N	5	10	4	L	..	49	40	C	1	5 15 10	4 4 8	L R	1	28
18	A	4	10	4	R	4	40	41	C	1	10	4	R-L	1	47
19	B	4	10	4	R	4	26	42	N	1	10 10	4 8	L R	1	25
20	C	4	30 10	8 4	R L	4	31	43	C	1	5	8	R	1	30
21	C	4	10	4	L-R	4	30	44	B	½	5 10	8 4	R L	1	26
22	A	3	20	8	R	1	25	45	B	½	10 30	4 4	R L	1	29
23	B	3	20	4	L-R	2	26	46	B	½	10	4	L-R	1	25

* Decibels lost in excess of the initially established threshold in the designated frequency.
† Year in which a significant change in the threshold was first stabilized and detected.

TABLE 7.—*Correlation of Duration and Intensity of Exposure with Audiometric Changes in Normal Group*

Years Studied	Exposure Group	Change +	Change −
5	A	..	2
	B	..	2
	C	1	4
	N	..	1
4	C	..	1
	N	..	2
3	A	..	1
	B	..	1
	C	..	5
	N	..	1
2	A	1	1
	B	2	2
	C	1	5
1	A	1	4
	B	..	3
	C	..	4
½	B	2	5
	C	1	8
Totals		9	50

None of the 59 subjects showed change in hearing acuity in the frequencies 1024 and 2048. About half the changes occurred at 4096 and half at 8192. All the changes were unilateral, and the maximum loss was 25 db. from the 0-threshold level. If the criterion for a significant change in threshold were 25 instead of 15 db. for normal ears, only two subjects could be considered to have experienced any loss in

TABLE 8.—*Correlation of Duration and Intensity of Exposure with Audiometric Change in Abnormal Group*

Years Studied	Exposure Group	Change	
		+	−
5	A	1	7
	B	10	6
	C	5	5
	N	1	..
4	A	1	..
	B	1	2
	C	2	1
3	A	1	..
	B	2	1
	C	..	3
2	A	1	1
	B	3	1
	C	4	2
	N	..	1
1	A	4	1
	B	2	4
	C	4	3
	N	1	..
½	A	..	3
	B	3	..
	C	..	8
Totals		46	49

TABLE 9.—*Correlation of Intensity of Exposure with Audiometric Changes*

Exposure Group	Normal		Abnormal	
	Changed	Unchanged	Changed	Unchanged
A	2	6	8	12
B	4	11	21	14
C	3	28	15	22
N	0	4	2	1
Total	9	50	46	49

hearing acuity during this study. It is too early to describe the progressive character of any of these hearing changes; however, there is no definite indication at present that any of them can be attributed to exposure to this specific type of occupational noise. It may develop that the hearing loss detected within the first year of exposure could be due to acoustic trauma, but it is of such a mild degree and so insidious as to be comparatively inconclusive in the present stage of this investigation.

Table 2 shows that 95 (62%) of the 154 subjects for whom audiograms were taken had significant losses of 10 db. or more in the higher frequencies when the initial hearing survey was made. During the investigation 46 of these subjects

(48%) showed further significant audiometric changes, or a minimum of a 10 db. loss in any frequency. Details, correlations, and summaries are presented in Tables 5, 6, 8, and 9.

Among the 49 subjects who showed no change in hearing were 4 who had the most intense exposure of all. These men were frequently exposed to after-burner noise for prolonged, intermittent periods. Subject 1, who also was exposed to after-burner noise and had considerably more than the minimum exposure in Group A, showed a slight loss bilaterally in one frequency. Eighteen subjects exposed for five years, and twelve subjects in Group A were among those not showing any significant audiometric change.

The 46 men who sustained hearing changes included 8 in Group A, 21 in Group B, and 15 in Group C. Two were in Group N without any exposure to intense noise. One-half of this group had been exposed to gunfire and half had not. Of the 46 subjects 24 were exposed to jet noise for three or more years. It is interesting to note in Column 8 of Table 6 that the first 21 subjects were a decade older than the subjects in the second half of the chart and that, correspondingly in Column 7, the hearing losses were detected after the third year in the older group but during the first or second year in the younger group. The year in which the hearing change was first detected should not be misconstrued as a precipitous alteration in hearing acuity. It merely indicates that only after that period of time did the change in threshold stabilize itself to fit into the specified criterion. Generally most of the hearing losses fluctuated before stabilizing at a level 5 db., 10 db., or more from the initially established threshold.

None of the subjects in this group showed audiometric change at 1024. Subject 33 was the only one to show a change at 2048, and this was only a 5-db. loss unaccompanied by change in any other frequency. Column 4, Table 6, reveals the hearing loss in decibels above the threshold established at the start of the investigation for each subject. If a 20-db. change instead of a 10-db. loss were the level of significance, only 17 subjects would fall into the group showing change in threshold.

There is no convincing evidence that as the intensity and duration of exposure increased within the limits described the incidence or degree of hearing loss became larger in the group of subjects evaluated in this study.

It can be shown, for instance, that of the 95 subjects with abnormal initial hearing thresholds 57 had previously been exposed to the acoustic trauma of small-arms gunfire as well as other industrial noise. The gunfire could well account for a large proportion of the hearing losses at 4000 and 8000 cycles. Of these 95 subjects 22 were over 40 years of age. An evaluation of the individual charts reveals that their hearing losses could be attributed to presbycusis.

In reference to the 38 subjects in this group who initially showed abnormal hearing but gave no history of exposure to any intense noise, it might be interesting to cite the important contribution made by Loch [32] in 1943, when he studied the incidence of tonal dips in 1,365 children between the ages of 8 and 14. On the basis of frequencies similar to those tested in the present investigation, Loch found that 9% of the children showed significant tonal dips, most of which occurred at 4096. Furthermore, he indicated that the incidence of tonal dips increased as the children grew older and that the incidence was much higher in males.

32. Loch, W. E.: Incidence and Permanency of Tonal Dips in Children, Laryngoscope **53**:347-356, 1943.

A critical appraisal of the chart of each subject with hearing change gives a marked impression that the insidious hearing changes could well be attributed to factors other than exposure to jet-engine noise. The general picture of the audiometric changes encountered is consistent with the clinical picture of presbycusis, and there is a strong likelihood that these changes would have occurred irrespective of the exposure to jet-engine noise. Much more investigation is indicated to confirm or contradict this impression. If the continuous noise described in this report or any reasonably similar noise has any effect at all upon hearing acuity, it is certainly not a sudden dramatic effect such as one sees in exposure to gunfire or chipping.

Tinnitus and subjective hearing impairment were rarely encountered in any of the subjects.

There is a strong inclination to compare the 15% of normal subjects whose hearing changed with the 48% of abnormal subjects whose hearing changed and to conclude that abnormal ears are more sensitive to noise than normal ears. I wish to emphasize that such a conclusion on the basis of the present data is inadvisable and probably incorrect. There even are many indications that within certain limits to be investigated in the future some ears with inner-ear hearing losses are less susceptible to acoustic trauma than some normal ears.

At present the cumulative effect of acoustic trauma is unconfirmed by experimental data. The problem basically needs further definition. By some it is interpreted as meaning that once a subject has suffered hearing damage be becomes more sensitive to further damage by noise of lesser intensity than when his hearing was normal. To others cumulative effect means that, although a certain noise may be insufficiently loud to produce immediate hearing damage, if the subject is exposed to the noise repeatedly or for a sufficient length of time the subclinical hearing loss will accumulate and become manifest on the audiogram. Each of these interpretations necessitates considerably more investigation and consideration.

SUMMARY

A review of the literature reveals considerable difference of opinion concerning the cumulative effect of noise upon hearing and divergent estimates of maximum safe intensity levels ranging from 75 to 120 db.

The object of the present investigation was to study the effect upon hearing acuity of prolonged exposure to a specific steady-state noise ranging in intensity between 90 and 120 db.

Comprehensive hearing studies were performed over an approximate five-year period on 272 subjects who were daily exposed to a specific type of jet-engine noise. The intensity and frequency characteristics of the noise and the duration of each subject's exposure were correlated with any significant changes in hearing.

The subjects used in this investigation were representative of a large number of trained specialists now being exposed to similar noises in many industries throughout the country.

In order to adhere to the most rigid criteria possible, only 154 of the 272 subjects are included in this preliminary report. Subjects with conductive-hearing loss, those using ear protectors, and those for whom complete data were not available were excluded.

Ambient-noise levels were evaluated, as was the effect of auditory fatigue, in order to obtain stabilized hearing thresholds.

CONCLUSIONS

As a result of data gathered in this preliminary report, the following conclusions are suggested.

There is considerable indication that a certain steady-state jet-engine noise with intensity fluctuations between 90 and 120 db. and with sound energy predominantly in the frequencies below 600 cycles is not hazardous to the hearing of exposed personnel.

Personnel exposed to steady-state moderately intense low-frequency noise may sustain measurable degrees of auditory fatigue daily for many years without sustaining any significant degree of permanent hearing impairment.

None of the subjects in this study who were exposed to moderatly intense low-frequency noise for periods up to five years showed any change in hearing acuity in the frequencies 1024 and 2048.

None of the subjects in this investigation sustained changes in hearing acuity greater than 30 db. at 4096 and 8192.

None of the changes in hearing occurring in any of the subjects in this study can definitely be attributed to exposure to jet-engine noise.

Subjective hearing loss and tinnitus were rarely encountered and were not related to the occupational noise being investigated.

From the observations made in this study thus far, the human ear would appear to be more resistant to injury by noise than it is usually thought to be.[33]

1721 Pine St.

33. Guild, S. R.: Personal communication to the author.

CALIBRATED WEBER TEST IN FENESTRATION SURGERY

Evaluation of Postoperative Serous Labyrinthitis

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POSTOPERATIVE serous labyrinthitis is one of the great problems in fenestration surgery today. To reduce or eliminate postoperative serous labyrinthitis, there must be a calibrated method of measuring its type, degree, and duration. To obtain this goal, we have calibrated the Weber test. By adapting the long-proved Weber test to the bone-conduction oscillator of the audiometer, it has been possible to demonstrate the variations in threshold and, with increasing intensity, variations in lateralization of sound. As presented, the calibrated Weber test may be described as a new hearing test; in actuality, it is only an audiometric adaptation of one of the oldest hearing tests. This test has been of value to us in determining the status and prognosis in our recent cases of fenestration. In summary, this article is the report of a study on the behavior of bone conduction in the recently fenestrated ear.

As stated, the calibrated Weber test will demonstrate variations in threshold and variations in lateralization. As will be described later, the threshold of bone conduction may be either improved by uncapping the cochlear reserve or depressed by trauma or serous labyrinthitis. The shift of lateralization of the sound with increasing intensity results from the hypersensitivity of the edematous postoperative ear, which is the phenomenon of recruitment. Since the preoperative bone conduction is usually equal in the two ears, the calibrated Weber test will, by comparison, demonstrate these findings without annoying the convalescent patient with masking noises.

This study is a direct outgrowth of the tuning-fork Weber test as performed by Juers [1] and the scientific exhibit on recruitment by Simonton.[2] In performing the postoperative Weber test of Juers, we noticed that lateralization might vary with repeated testing. If we performed the Weber test with a light tap of the

From the Southern Foundation of Better Hearing, Tampa Municipal Hospital.

Presented as a scientific exhibit at the American Academy of Ophthalmology and Otolaryngology, Chicago, October, 1952.

1. Juers, A. L.: Observations on Bone Conduction in Fenestration Cases: Physiological Considerations, Ann. Oto. Rhin. & Laryng. **57**:28-40 (March) 1948.

2. Simonton, K. M.: Diplacusis Binauralis Dysharmonica and Recruitment of Loudness: Their clinical significance, Scientific Exhibit on Recruitment, American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 14-19, 1951.

tuning fork, the sound would frequently lateralize to the ear not operated upon. This shift of lateralization would cause us great mental depression because we felt that it indicated the possibility of a poor end-result. However, we would immediately strike the tuning fork a heavier blow and repeat the Webr test, and with the increased intensity of sound there would be a shift of lateralization back to the ear operated upon, immediately making us happy about the condition of our patient. Wondering about the whys and wherefores of these variations, we began to perform the calibrated Weber test with the audiometer. With the calibrated Weber test, we found it possible to demonstrate recruitment by bone conduction in a manner similar to the binaural loudness balance test of air conduction (Fowler [3]).

The resulting detailed information gave us some insight into the postoperative behavior of the inner ear. The calibrated Weber test is time consuming and therefore will never be popular. It will be of value in research studies on variations in technique. Knowledge of the findings and interpretations will, however, be of great value in proper interpretation of the simple tuning-fork Weber test.

For clarity, the study will be presented, with some repetition, in the following order:

1. A schematic presentation of the postoperative course of the bone-conduction threshold.
2. The technique of the calibrated Weber test.
3. The classification of lateralization.
4. The correlation of this study with the end-results for (*a*) primary lateralization to the ear operated upon, (*b*) the secondary variations in lateralization, (*c*) the effect on high tones, and (*d*) the tertiary bone-conduction threshold.
5. Review of preexisting and concomitant studies.
6. Comments and summary.

POSTOPERATIVE COURSE OF BONE-CONDUCTION THRESHOLD

The usual postoperative course of the bone-conduction threshold may be divided into three phases: primary improvement, secondary depression, and tertiary improvement (Fig. 1).

In our studies and in the studies of Juers, Shambaugh, and others, we frequently noticed an improvement in the threshold of bone conduction immediately after fenestration surgery. This primary improvement is believed to result from the improved mobility of the perilymph, the improved mobility of the perilymph permitting greater transmission of sound to the end-organ. The phenomenon of primary improvement in bone conduction would not be observed if there was round-window fixation or inadequate cochlear reserve. Significant surgical trauma to the membranous labyrinth or operative hemorrhage into the inner ear would, on the other hand, produce a primary depression in bone conduction. Such primary depression has been called serous labyrinthitis, although in actuality it is an immediate surgical injury without the phases of secondary inflammatory reaction and would be better

3. Fowler, E. P.: Marked Deafened Areas in Normal Ears, Arch. Otolaryng. **8**:151-155 (Aug.) 1928; The Diagnosis of Diseases of the Neural Mechanism by the Aid of Sounds Well Above Threshold, Tr. Am. Laryng. Rhin. & Otol. Soc. **41**:207-219, 1937; The Recruitment of Loudness Phenomenon, Laryngoscope **60**:680-695 (July) 1950.

named primary surgical depression. For uniformity, we will classify it as primary surgical labyrinthitis, demonstrable on the first postoperative day.

The secondary depression in bone-conduction threshold is the manifestation of true serous labyrinthitis, a sterile inflammatory reaction. This inflammatory reaction begins with the surgical stimulus, is of gradual onset, and usually reaches its maximum on the seventh to ninth postoperative day. The degree of serous labyrinthitis is dependent upon inflammatory edema within the inner ear. This inflammatory edema varies with the degree and type of surgical trauma. Day [4] has pointed out that the degree of postoperative serous labyrinthitis is directly dependent upon the degree of trauma to the flap. A traumatized flap will produce maximum dilatation of the vessels in the flap, maximal exudate into the inner ear, and a maximum degree of serous labyrinthitis and postoperative depression in hearing. Lamp [5] and his co-workers have pointed out that the perilymph and endolymph are slightly hypertonic to isotonic sodium chloride solution; therefore, they have reasoned

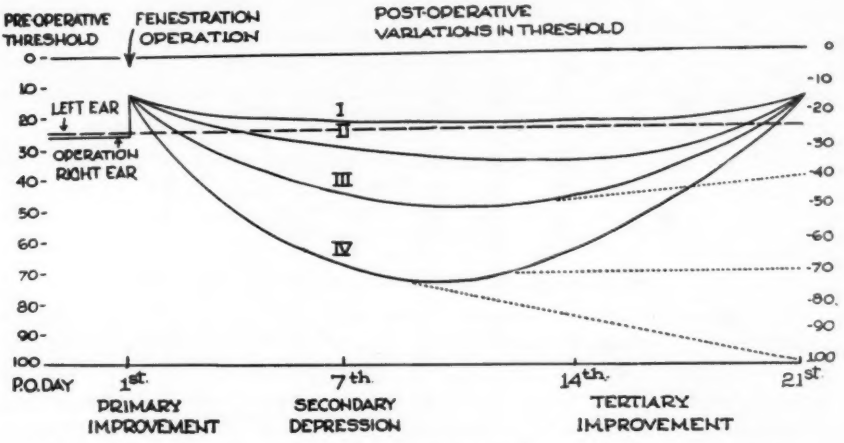

Fig. 1.—Postoperative course of bone conduction threshold may be divided into three phases: primary improvement, secondary depression, and tertiary improvement. This schematic presentation demonstrates the usual variations in threshold.

that irrigation with isotonic sodium chloride solution would result in retained fluid within the inner ear, this osmotic increment producing a labyrinthine hydrops and depression of hearing. Lamp,[5] therefore, recommends irrigation with solution isotonic to the perilymph. The trauma of irrigation and manipulation of the endosteum, perilymph, and membranous labyrinth are stimuli resulting in serous labyrinthitis and postoperative depression of hearing. The degree of serous labyrinthitis will vary with the severity of the stimulus and may be classified as minimal, mild, moderate, or severe postoperative serous labyrinthitis (Curves I, II, III, and IV, Fig. 1).

4. Day, K. M.: Appraisal of Fenestration Operation: Report of 100 Cases, Arch. Otolaryng. **44**:547-559 (Nov.) 1946.

5. Lamp, C. B., Jr.: Unpublished data, presented to the Otosclerosis Study Group, Chicago, 1952.

The third phase in the postoperative course of the bone-conduction threshold is tertiary improvemnt. Both the primary surgical depression and the secondary serous labyrinthitis may be and usually are reversible processes; therefore, in most instances the inner ear completely recovers and bone conduction returns to the preoperative level or above. As a rule, the recovery occurs in the third postoperative week. The length of the recovery period and the degree of permanent loss are directly proportional to the degree of surgical trauma and the resultant serous labyrinthitis.

The immediate evidences of irreparable damage by serous labyrinthitis are, in mild cases, loss of high tones and, in more severe cases, unsatisfactory return of useful hearing. Later, there may be a degenerative terminal nerve deafness.

On the basis of these observations, the sterile postoperative reactions within the inner ear may be classified as follows:

I. Primary surgical labyrinthitis
  A. Surgical trauma
  B. Operative hemorrhage
II. Secondary serous labyrinthitis
  A. Minimal serous labyrinthitis
  B. Mild serous labyrinthitis
  C. Moderate serous labyrinthitis
  D. Severe serous labyrinthitis
III. Tertiary cicatricial labyrinthitis
  A. Loss of high tones
  B. Unsatisfactory recovery
  C. Terminal nerve deafness

TECHNIQUE OF THE CALIBRATED WEBER TEST

The calibrated Weber test is performed by placing the bone-conduction oscillator in the center of the forehead and testing with intermittent ascending stimulation for the frequencies of 500, 1,000, and 2,000 cps. With each frequency, the threshold of bone conduction is first determined; the lateralization of bone conduction is then determined at 10-db. steps to the maximum, 60-db., level. The test is recorded on the routine audiogram form (Fig. 2), routine markers being used to indicate lateralization to the right or left or both ears. Figure 2 is a theoretical and improbable audiogram to show the recording of lateralization to the left ear, or the ear not operated on, at the frequency of 500 cps. At 1,000 cps lateralization was first to the left ear, or the ear not operatd on; then it was to both ears, and finally, there was a shift of lateralization back to the right ear, the ear operated on, with the maximum intensity. At 2,000 cps there was complete lateralization to the ear operated on, with some improvement in threshold.

Preoperatively, the patient is tested and carefully instructed.

In this detailed and basic study, tests were performed daily for the first eight postoperative days and then at semiweekly intervals until the hearing became stabilized. For practical application, we recommend the calibrated Weber test on the 1st, 7th, 14th, and 21st postoperative days. Testing on the first postoperative day is most important to demonstrate the primary response; if it is inconvenient to do the complete calibrated Weber test on this day, the tuning-fork test will suffice, for there has been no time for recruitment to develop with shifting lateralization by variation in intensity.

CLASSIFICATION OF LATERALIZATION

In the calibrated Weber test, lateralization has been found to vary from complete lateralization to the ear operated on (ipsilateralization) to complete lateralization to the ear not operated on (contralateralization). In this study, we recorded our findings with the following classification:

I. *Ipsilateralization* (Fig. 3).—In ipsilateralization, there is complete lateralization to the ear operated on from threshold to maximum intensity. Figure 3 illustrates this lateralization at intensities of 10, 20, 30, 40, 50, and 60 db. Figure 1 illustrates an improved threshold for bone conduction, for in the ear not operated on the threshold for the calibrated Weber test is usually at or near the 15-to-25 db. level. In otosclerosis the stapedial fixation seals the perilabyrinthine space and inhibits vibration of the perilymph. When the new fenestra is created, there is improved

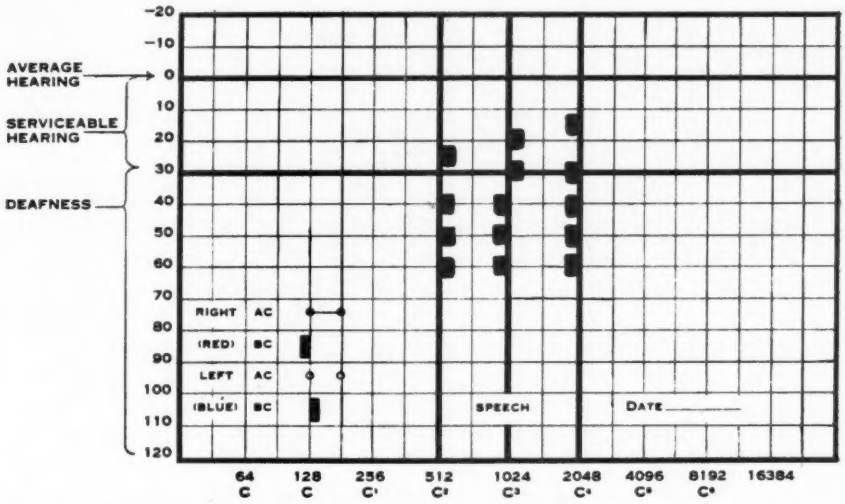

Fig. 2.—Theoretical audiogram demonstrating method of recording lateralization in calibrated Weber test. Bone-conduction markers indicate that sound lateralized to right, left, or both ears at recorded intensity.

mobility of the perilymph, improved transmission of sound to the end-organ, and, clinically, an improved bone-conduction threshold. The improved bone-conduction threshold results in complete lateralization to the ear operated on—ipsilateralization.

II. *Bilateralization* (Fig. 4).—Lateralization to both ears (failure to lateralize) occurs when bone conduction is the same in the two ears and when there is no recruitment. In the recently fenestrated ear, bilateralization would occur when there was failure of the threshold to improve, as in the case of round-window fixation or operative hemorrhage into the inner ear. A mild postoperative depression in hearing would result in bilateralization.

III. *Partial Contralateralization* (Fig. 5).—The term partial contralateralization is used to describe a partial shift of lateralization to the ear not operated on. Figure 5 illustrates lateralization to the left ear, the ear not operated on, at the 20-db. level; lateralization to both ears at 30 and 40 db., and a shift of lateralization

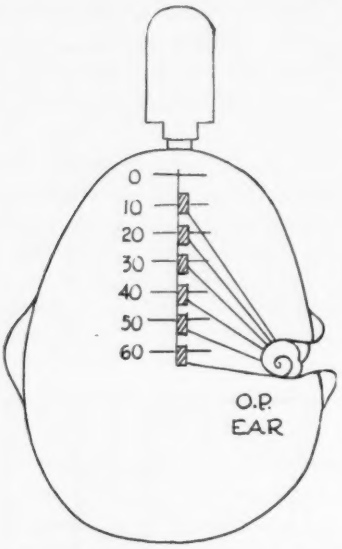

Fig. 3.—Ipsilateralization. Bone-conduction oscillator is placed in center of forehead in calibrated Weber test. This figure illustrates complete lateralization to ear operated on in 10-db. steps from threshold at 10-db. level through maximum intensity at 60 db. Ipsilateralization is defined as complete lateralization to ear operated on at all intensities.

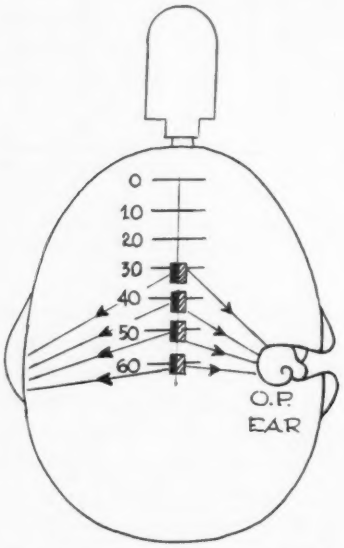

Fig. 4.—Bilateralization. Lateralization to both ears at intensities of 30, 40, 50 and 60 db. Bilateralization is defined as lateralization to both ears at all intensities.

to the ear operated on at 50 and 60 db. This finding indicates serous labyrinthitis with a depression in threshold but increased sensitivity to greater volume. The sound is, therefore, heard first in the ear not operated on, then in both ears, and then in the ear operated on. The calibrated Weber test here demonstrates the phenomenon of recruitment in a manner similar to that of the binaural loudness balance test of air-conduction audiometry.

We have observed this phenomenon in repeated instances. In our classification, there is lateralization to the ear operated on at the maximum intensity, resulting from a pathological depression in threshold and a pathological sensitivity to loud sound. This pathological lateralization to the ear operated on differs entirely from physiological lateralization to the ear operated on, which is due to an improved threshold, as illustrated in Figure 3. Lateralization to the ear operated on due to undesirable recruitment and lateralization to this ear due to a desirable improved threshold cannot be accurately differentiated with the tuning-fork Weber test.

IV. *Complete Contralateralization* (Fig. 6).—The term complete contralateralization describes complete lateralization to the ear not operated on from threshold to the maximum intensity of the audiometer. This finding indicates a profound depression of function in the ear operated on.

CORRELATION OF FINDINGS

With the calibrated Weber test, in the usual postoperative course we recorded the three phases: the phase of improvement with primary ipsilateralization, the phase of secondary depression with secondary variations in lateralization, and the phase of tertiary improvement with return of ipsilateralization and improved threshold.

The interpretations presented are based on 726 calibrated Weber tests on 64 patients who had undergone fenestration operations. The statistics present an analysis of 40 cases with preoperatively matched bone conduction. Preoperatively, the otosclerotic patient usually showed bilateralization at frequencies of 500 and 1,000 cps but lateralization to the better ear at 2,000 cps.

The study was conducted under office-practice circumstances in a sound-treated room with a Maico H-1 audiometer. The patient was transported to the hearing room daily by stretcher or wheelchair. In the immediate postoperative period, the reliability of testing is sometimes altered by the patient's condition. With due appreciation for this fact and for the limitations of the Weber test, we believe that the group trends of this small series and the interpretations will be substantiated when the study is repeated by a psychoacoustic laboratory.

Primary Ipsilateralization.—Primary ipsilateralization was observed on the first postoperative day in 75% of cases. Of the 30 patients who showed primary ipsilateralization, 100% ultimately obtained a satisfactory restoration of hearing. Of the 10 who did not show primary ipsilateralization, only 80% ultimately obtained a satisfactory restoration of hearing. Of the two who did not show primary ipsilateralization and did not obtain a satisfactory restoration of hearing, one had a known injury to the membranous labyrinth. On the basis of these findings, primary ipsilateralization appears significant in prognosticating the ultimate end-result and appears more important than secondary variations in lateralization. As previously stated, failure to obtain primary ipsilateralization may result from inadequate

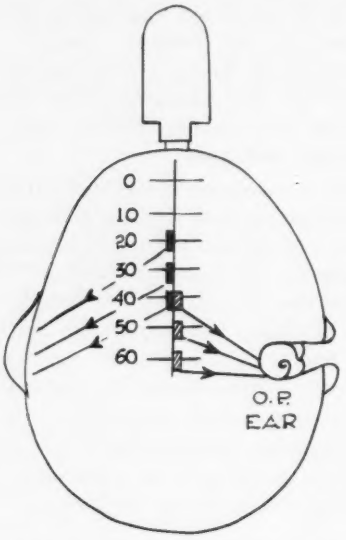

Fig. 5.—Partial contralaterilization. Shift of lateralization to ear not operated on at 20- and 30-db. levels, bilateralization at 40-db. level, and return of lateralization to ear operated on at 50- and 60-db. levels. This is phenomenon of recruitment demonstrated by bone conduction. Partial contralateralization is defined as lateralization to ear not operated on at threshold, with return of lateralization to ear operated on with increasing intensity.

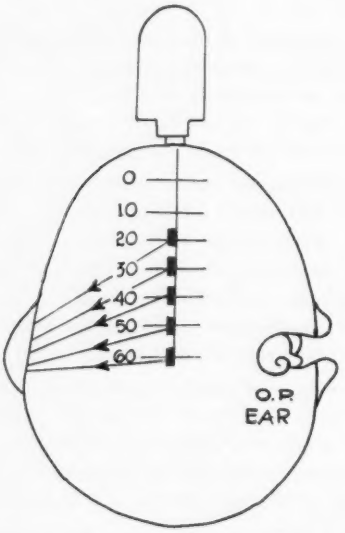

Fig. 6.—Complete contralateralization to ear not operated on at all intensities from threshhold of 20 db. to maximum intensity of 60 db., demonstrating profound depression in bone conduction in ear operated on. Complete contralateralization is defined as complete lateralization to ear not operated on at all intensities.

cochlear reserve, from round-window fixation, or from immediate surgical trauma to the membranous labyrinth or immediate operative hemorrhage. Failure to lateralize may, in some instances, be influenced by the patient's inability to cooperate on the first postoperative day.

It is our opinion that the cupola technique of Lempert [6] will yield a higher incidence of primary ipsilateralization than will operation in which the window is made under irrigation. However, we feel that sharp, hollow-ground margins of the fenestra made under continuous irrigation give a greater opportunity for primary healing of the margins and permanent patency of the fenestra.

Secondary Variations in Lateralization.—Secondary variations in the lateralization of bone conduction are produced by the sterile postoperative inflammatory reaction called serous labyrinthitis, here classified as minimal (I), mild (II), moderate (III), and severe (IV) (Fig. 1, Curves I, II, III, and IV). Minimal serous labyrinthitis was classified as that in which there was sustained lateralization to the ear operated on throughout the postoperative course. Although the patient may have shown secondary depression in the threshold of bone conduction, this depression was never great enough to cause a shift of lateralization to the ear not operated on. Mild serous labyrinthitis was classified as that in which there was enough secondary depression for the patient to lose the primary ipsilateralization and to show bilateralization but not sufficient depression for him to show contralateralization. Moderate serous labyrinthitis was classified as that in which there was sufficient secondary depression of bone conduction for the patient to show partial contralateralization. Severe serous labyrinthitis was classified as that in which the patient showed profound depression in hearing and complete shift of lateralization to the ear not operated on.

Of the 40 patients whose cases were analyzed, 17.5% showed sustained ipsilateralization through the postoperative course and were classified as having minimal serous labyrinthitis. These patients had a preoperative average hearing level of 47 db. and a postoperative level of 21.4 db.

Thirty-five per cent of the patients showed mild serous labyrinthitis, with bilateralization at the time of greatest depression; these patients had a preoperative average hearing level of 52.7 db. and a postoperative average of 25.1 db.

Forty-five per cent of the patients showed moderate serous labyrinthitis, with partial contralateralization, in the calibrated Weber test. These patients had an average preoperative hearing level of 50.4 db. and an average postoperative hearing level of 25.4 db. With the tuning-fork tests, Groups I and III, whom we classified as having minimal and moderate postoperative serous labyrinthitis, would have fallen into the same group, for both showed lateralization to the ear operated on at maximum intensity.

Two and five-tenths per cent of the patients showed complete shift of lateralization to the ear not operated on, with complete loss of hearing.

Effect on High Tones.—The average decibel gain in Groups I, II, and III showed no great variation. Group I showed 26.6 db.; Group II, 27.6 db., and Group III, 25 db. More interesting and probably of greater significance is the effect

6. Lempert, J.: Bone-Dust-Free Lempert Fenestra Nov-Ovalis: New Evolutionary Development of Surgical Treatment of Clinical Otosclerosis, Arch. Otolaryng. **47**:280-288 (March) 1948.

of the degree of serous labyrinthitis upon tones of 4,000 and 8,000 cps. Of those 30 patients who preoperatively had flat or ascending audiograms, the 16.6% who had sustained ipsilateralization showed 100% maintained hearing for the higher tones. The 33.3% who had bilateralization, or minimal serous labyrinthitis, showed 80% maintained hearing for the high tones. The 50% who had partial contralateralization, or moderate serous labyrinthitis, showed only 60% maintained hearing for the high tones. These figures being reversed, there was no loss, 20% loss, or 40% loss of high tones, according to the degree of serous labyrinthitis. The loss for high tones has long been regarded as a result of the serous labyrinthitis. This small series would seem to indicate a direct correlation between the variation in lateralization as demonstrated by the calibrated Weber test and the loss of the higher tones.

Tertiary Bone-Conduction Threshold.—With the calibrated Weber test, we were able to demonstrate tertiary improvement in bone conduction. For the three speech frequencies, there was an average improvement of 12.9 db., the greatest improvement being at the frequency of 2,000 cps. As the testing progressed, we became more and more interested in the bone-conduction threshold, and the patients became more and more accustomed to the testing. These facts might account for a few decibels' exaggeration in the actual improvement; however, we are convinced that there was an actual improvement in bone conduction, with a true figure of approximately 10 db. for the speech frequencies. Our studies substantiate the reports of Shambaugh and Carhart [7] and Woods.[8]

BILATERAL FENESTRATIONS

When a fenestration operation has previously been performed on the contralateral ear, there are two variables, the newly fenestrated ear and the formerly fenestrated ear. Of the 64 cases, 14 were not included in the figures presented because in 7 the ears were unmatched and in 7 the fenestrations were bilateral. A few general impressions have been gained from the bilateral fenestrations.

The Weber test will lateralize to the ear newly operated on in frequencies at which the threshold of bone conduction is above that of the primary ear. With depression of threshold, the Weber test will lateralize to the contralateral ear even though masking of the contralateral ear shows the depression of threshold to be only 10 db. On the first postoperative day, we saw in two instances lateralization to the ear newly operated on at threshold and shift of lateralization to the contralateral ear with increasing intensity. Only in cases of bilateral fenestration have we observed shifts with variations of intensity on the first postoperative day.

CORRELATED STUDIES

Modern hearing tests and modern knowledge of the interpretation of older hearing tests are largely the result of the stimulus provided by fenestration surgery. Prior to the era of such surgery, tuning-fork tests were largely performed with the 128 cps, the 256 cps, and, sometimes, the 512 cps fork. Lempert deserves credit

7. Shambaugh, G. E., Jr., and Carhart, R.: Contributions of Audiology to Fenestration Surgery; Including a Formula for the Precise Prediction of the Hearing Result, A. M. A. Arch. Otolaryng. **54**:699-712 (Dec.) 1951.

8. Woods, R. R.: Bone Conduction in Otosclerosis, Arch. Otolaryng. **51**:485-499 (April) 1950.

for introduction of the practical use of the 1,024-cps and 2,048-cps forks. The Weber test with the 2,048-cps fork is important in the preoperative selection of cases for two reasons: (1) if the patient cannot hear the 2,048-cps fork in the central position, he is a poor candidate for surgery and (2) in the central position, the dominant air conduction does not greatly interfere with the testing of true bone conduction. (This idea was taught to one of us [J. B. F.] by Maxwell.[9] but is believed to have originated with Lempert.) As a result of these preoperative studies, the tests were applied to the postoperative period.

Juers utilized the tuning-fork Weber test to evaluate the status of the postoperative fenestrated ear. He observed lateralization to the ear operated on in the first few postoperative days in 51 of 88 consecutive cases, or 57.9%. In the hospital period, he observed sustained lateralization to the ear operated on in 69 cases (this would include our Groups I and III, who, respectively, showed lateralization to the ear operated on due to improved threshold and to recruitment). In analysis of the six-week results with 15 db. or more gain, Juers reports sustained ipsilateralization in 64 cases, shift or partial return of lateralization in 10, and no return of lateralization in 3. In analysis of the six-week results showing less than 15 db. gain, he reports no shift in the Weber test in five and shift with no return in six. In testing bone-conduction threshold, Juers observed improved bone-conduction threshold immediately after operation and again three months postoperatively.

Juers has stressed use of the 512-cps and 1,024-cps forks. We feel that the frequency of 2,000 cps is equally or more important, for this is the frequency at which the greatest improvement in threshold is shown and the frequency for which hearing is most easily damaged by serous labyrinthitis. Further, with the decreasing intensity of tuning-fork sound, it is difficult to demonstrate shifts of lateralization. These findings are frequently demonstrated with the more accurate intermittent ascending stimulation of the calibrated Weber test.

Shambaugh and Carhart,[10] in studies of bone-conduction threshold in 150 consecutive cases of fenestration, also observed an immediate postoperative improvement of 10 to 15 db. In describing serous labyrinthitis, Shambaugh states:

> Between the fourth and the seventh day after operation, about half the cases experience a depression in bone conducted hearing, generally more pronounced for the higher frequencies. After a few days the bone conducted hearing in most of these then begins to recover, until several weeks or even months after operation, it finally reaches the immediate post-operative level. The bone conducted hearing for the higher frequencies is the last to recover, and in some cases it may never recover to the pre-operative level . . . in severe serous labyrinthitis.

In the usual good result, the final improvement in bone conduction from improved mobility of the perilymph is the basis of the "Carhart notch." McConnell and Carhart [11] have shown that fenestration surgery produces a real improvement in responsiveness to bone stimuli through the range from 1,000 through 4,000 cps.

9. Maxwell, J. H.: Personal instruction to one of the authors (J. B. F.).

10. Shambaugh, G. E., Jr.: Post-Operative Serous Labyrinthritis: Experimental and Clinical Studies, in Fenestration Operation for Otosclerosis, Acta oto-laryng., 1949, Supp. 79, Chap. 5, pp. 45 and 48; Correlation of the Predicted with the Actual Result of Fenestration in 164 Consecutive Cases, Tr. Am. Laryng. Rhin. & Otol. Soc. **56**:313-329, 1952. Shambaugh and Carhart.[7]

11. McConnell, F., and Carhart, R.: Influence of Fenestration Surgery on Bone Conduction Measurements, Laryngoscope **62**:1267-1291 (Dec.) 1952.

Woods [8] has done an excellent study on postoperative bone-conduction threshold and records an improvement of 11 db. for the speech frequencies. He states:

> In otosclerosis, the first air conduction loss is in the low tones while the first bone conduction loss is in the high tones. . . . It seems reasonable to suppose that fixation of the stapes not only prevents vibration from reaching the labyrinth but also impedes the response of the end-organ by limiting the mobility of the perilymph.

Henner [12] studied the threshold of bone conduction with the audiometric Weber test and in cases suitable for surgery found the midline threshold of 10 db. for 512 cps, 15 db. for 1,024 cps, 25 db. for 2,048 cps, and 15 to 20 db. for 4,096 cps.

Fowler [3] has described recruitment as relative sensitivity to increasing intensity of sound. His studies have been amplified and calibrated by Dix, Hallpike, and Hood.[13]

In the period during which we conducted our study, Markle, Fowler, and Moulonguet [14] conducted an independent parallel study utilizing an audiometric Weber test in the selection of ears requiring masking. In their article, they state, "Changes in lateralization, produced by changes in intensity of the test-tone, may indicate the presence of recruitment in the poorer ear."

In our study, we have correlated this preexisting knowledge into a test which has given us knowledge of the behavior of the postoperative fenestrated ear. Independently, we have developed the calibrated Weber test as a method of demonstrating recruitment by bone conduction.

SUMMARY

The postoperative course of bone-conduction threshold is described as showing three phases: primary improvement, secondary depression, and tertiary improvement.

The technique of the calibrated Weber test is described.

With the Weber test, the sound may lateralize to the ear operated on from two different causes: (1) desirable physiological improvement in threshold or (2) undesirable pathological recruitment.

The calibrated Weber test will differentiate lateralization to the ear operated on due to improved threshold from lateralization to the ear operated on due to through recruitment.

There is direct correlation between the secondary variations in the calibrated Weber test and the tertiary loss of high tones.

A tertiary improvement in bone-conduction threshold has been demonstrated by the calibrated Weber test.

Current related studies have been reviewed.

Knowledge of these findings will be of practical value in interpretation of the postoperative behavior of the fenestrated ear. It is hoped that the calibrated Weber

12. Henner, R.: Personal communication to the authors.

13. Dix, M. R.; Hallpike, C. S, and Hood, J D.: Observations upon the Loudness Recruitment Phenomenon with Especial Reference to the Differential Diagnosis of Disorders of the Internal Ear, and Eighth Nerve, J. Laryng. & Otol. **62**:671-686 (Nov.) 1948.

14. Markle, D. M.; Fowler, E. P., Jr., and Moulonguet, H.: The Audiometer Weber Test as a Means of Determining the Need for, and Type of, Masking, Ann. Otol. Rhin. & Laryng. **61**:888-900 (Sept.) 1952.

test will prove of academic value in the detailed study of postoperative serous labyrinthitis.

Current investigations in fenestration surgery are designed to reduce postoperative serous labyrinthitis. Variations in technique cannot be accurately evaluated by clinical impressions. For the evaluation of postoperative serous labyrinthitis, the following criteria are proposed:

1. The presence or absence of primary improvement.
2. The degree of secondary variations in bone conduction as manifested by shifts of lateralization, depression of threshold, diplacusis, or recruitment.
3. The time and degree of tertiary improvement in bone conduction.
4. The six-week improvement in air conduction for high tones and for the usual speech frequencies.
5. The incidence of terminal nerve deafness.

ORIENTATION IN THERAPEUTICS OF PAPILLOMA OF LARYNX

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PAPILLOMA of the larynx in adults is a problem very difficult of solution. In the child it commonly disappears when puberty arrives. Several treatments, medical (as the use of calcined magnesia), physical, and surgical are recognized. Disappearance of papilloma at puberty makes us think of the possibility of a local action of estrogens. However, all therapeutic attempts using estrogens have failed. My colleagues and I also have had opportunity to apply this method, without satisfactory results. We commonly recommend vocal rest and the use of calcined magnesia when the patient remains under our control. Removal of the vilosities is done only to facilitate respiration. We do not agree with the use of radiotherapy because of its inefficacy, noticed on several occasions. It is important to note that several authors, among them Spira, consider a grave professional fault the failure to use this therapy, especially in the child. Vocal rest in the child is obtained by tracheotomy; that is what we always advise when the patient cannot remain under our observation. This operation is useful both in maintaining the patency of the respiratory tract and in treating the papilloma. It is a short-term therapeutic procedure; the completion of therapy must wait for the arrival of puberty.

In the adult the papilloma develops in an unpredictable way. In some patients simple removal is sufficient for a cure; in others the papilloma recurs after several interventions. We have no method for appraising how the papilloma will develop in the adult. There are not uncommon cases of malignant degeneration. Dealing with a benign tumor whose development cannot be forseen, we have no basis for adopting from the beginning a radical therapy.

Wan Wildenberger thinks there are two kinds of papillomas: those which are cured and those which recur. He is induced to believe not in two kinds of tumors, but in two ways of evolution of the same affection. At present some authors place this tumor in a special category between the benign and malignant ones. Salomon Balsa designates them as papilloepithelioma.

There are problems concerning papillomas that deserve consideration. Do papillomas predispose the tissue for evolution of the malignant tumor? Do the two processes develop side by side? Is there malignant degeneration of the papilloma? Each of the above-mentioned cases is referred to in the literature. Published observations show cases in which papillomas were removed several times; every case was controlled by pathological examination in which only several years afterwards malignancy was found. The validity of these examinations is beyond any doubt, for it was always possible to examine the whole tumoral mass. In other observations superficial biopsies are mentioned; when they are performed in deeper planes, the pathological structure differs from that found when superficial

Read before the Brazilian College of Surgeons, Rio de Janeiro.

planes are examined. It is not possible in this paper to go into particular details about a matter discussed so much.

In the adult we would rather use the indirect method of laryngoscopy for exeresis of papillomas and to perform biopsies. Dealing with papilloma in this way is very important, since these patients must endure several interventions. For the patient the difference between the direct and indirect route is very important; the indirect way is an ambulatory procedure which does not need great preparation and is neither annoying nor painful, being easily done. We think that the direct ways is used only by those who have not had enough practice in treating this condition. We recognize the indications for its use; the technique of execution of such interventions in the adult is, moreover, indispensable in treating the child.

When the papilloma involves only one vocal cord, its extirpation by laryngofissure with electrosurgery seems to be the best treatment. When the papillomas are located in both vocal cords, the indication for radical treatment is doubtful, since we have no basis for performing laryngectomy. We have already had opportunity to follow two patients in whom several pathology examinations showed only papilloma, but in which espinocellular epithelioma developed after some years.

We also had patients whose cures were achieved by simple extirpation of the papilloma. In any case, whenever we diagnose a papilloma, whether diffuse or localized, we must observe the patient for a long time. Such observation must be made even of those who are apparently cured. There are several histories of patients whose clinical cure was achieved for two, three, and even more years, but later malignant development was observed. In the literature we find cases in which there was a latent period of 7, 10 and even 25 years (Burth). It is believed that development of cancer of the larynx does take a period longer than three years, but it is difficult to know just when degeneration begins.

We try to remove the whole tumor mass when we perform the biopsy, but that is possible only when the neoformation is localized. We wait then for the pathological examination and examine the history of the case to determine the therapy; in the case of a localized tumor or even when both vocal cords are involved diathermocoagulation by laryngofissure is indicated. When the neoformation extends to the whole larynx it is necessary to be careful and obtain a better knowledge of the case, since on certain occasions laryngectomy may be required. In either eventuality we advise postoperative x-ray treatment. We agree with those who say that x-ray treatment is more useful in preventing recurrences than in dissolving the papillomas. There are other treatments whose use was formerly recognized, such as intubation, automatic curettage, and the use of local caustics, such as salicylic acid, chromic acid, or silver nitrate. There is even the possibility of spontaneous disappearance of the papillomas after an emotional shock, just as sometimes occurs with warts.

We are essaying vaccinotherapy in the adult and the use of podophyllin solution in the child. The results of these investigations will be presented at a later time.

We have made these comments about a matter that has interested us for a long time, with the desire to hear more authorized opinions not only from otorhinolaryngologists but also from those in other specialties in which these problems also appear.

Rua Santa Clara 369.

Case Reports

OPERATIVE AND POSTOPERATIVE MANAGEMENT OF BILATERAL BONY ATRESIA OF THE CHOANA

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BILATERAL bony atresia of the choana is encountered so infrequently that its diagnosis is often overlooked. The purpose of this paper is to suggest a simple surgical method of managing the choana patent postoperatively and preventing atresia. The subject of choanal obstruction has been so extensively discussed by Beinfield [1] and Dolowitz and Holley [2] in recent articles that I shall omit extensive bibliography and discussion of anatomy, embryology, and etiology. I shall concentrate on reporting the history of a patient who had a congenital bilateral osseous choanal obstruction, the simple operative procedure for its removal, and the attempt to restore patency and normal nasal respiratory physiology.

Three modes of operative approach were considered for the surgical treatment of the obstruction of both posterior nasal orifices: (1) transpalatal; (2) transseptal, by performing a submucous resection of the septum, including the perpendicular plate of the ethmoid, and removing the posterior, or distal, portion of the septum and the osseous obstruction laterally, and (3) limitation of surgery to the bony bilateral choanal obstruction.

After due consideration I found it most feasible to limit the surgery to removal of the bony choanal obstruction, thus preserving the nasal septum, palate, and mucous membrane.

REPORT OF A CASE

On Sept. 22, 1943, the child was delivered by the late Dr. D. S. Ward at the Suburban General Hospital, Pittsburgh. At birth her weight was 5 lb. 13 oz. (2,637 Gm.). An examination by Dr. Ward at that time revealed some form of nasal obstruction with profuse bilateral watery secretions. Otherwise the infant appeared to be normal. Twenty-four hours after birth convulsive seizures developed and were controlled by calcium administered intramuscularly. According to the records, there was difficulty in feeding, and there was vomiting after each feeding. A diagnosis of pyloric spasm was made at that time. On Sept. 25, after feeding a small amount of barium sulfate to the infant, an x-ray of the upper gastrointestinal tract was made in an attempt to determine whether an obstruction was present. Three hours after the feeding none of the meal had passed through the pylorus, and a film taken after five hours showed that no barium had passed through that area. After the various studies had been completed a provisional diagnosis of possible pyloric stenosis was made. A medical regimen

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1. Beinfield, H. H.: Treatment of Complete Unilateral Bony Atresia of the Posterior Nares: New Technique and Brief Reference to Asphyxia Neonatorum, A. M. A. Arch. Otolaryng. **53**:530-539 (May) 1951.

2. Dolowitz, D. A., and Holley, E. B.: Congenital Choanal Atresia: New Transpalatine Technic, Arch. Otolaryng. **49**:587-593 (June) 1949.

was started of atropine and phenobarbital with each feeding, and the vomiting subsided. All gastric symptoms disappeared during the next few days. The infant was able to retain the formula and did not show any further loss in weight.

She continued to be cyanotic and often became almost asphyxiated owing to the aforementioned impairment in nasal respiration. On Sept. 25, x-rays were taken of the thymus and sinuses by the late Dr. F. L. Schumacher. That of the thymus showed no thymic enlargement or tracheal compression. The sinuses were found to be undeveloped, and examination was therefore negative. The child progressed normally after a fashion and was discharged from the hospital nine weeks after birth (October 1943). She seemed to retain the formula, and her weight upon discharge was 5 lb. 15 oz. (2,693 Gm.), showing a gain of only 2 oz. (56.7 Gm.). The obstetrician advised the family that constant observation of the child was important and outlined the dangers and complications that were apt to occur because of the impaired nasal breathing.

Great care and vigilance were necessary to see that the child breathed properly through the mouth. On several occasions she was almost asphyxiated. At first, it was thought that she was a "blue baby," but her family physician soon eliminated the diagnosis of cardiac cyanosis by pulling the tongue outward in order to improve oral breathing. He ascribed the symptoms as being due to nasal obstruction rather than cardiac asphyxia. Air vents were inserted into the mouth, and these ameliorated the respiratory distress. The child probably owes her life to the fact that she had some pyloric symptoms and convulsions which necessitated special, undivided attention.

During the interim between infancy and early childhood, she had recurrent sore throat and enlarged tonsils. At the age of 5 there developed a cervical adenitis and dysphagia, which were attributed to hypertrophied tonsils and adenoids. The child was treated with antibiotics, and a tonsillectomy and adenoidectomy were performed at the Suburban General Hospital on Oct. 19, 1950. Once again, a complete study of the nasal pathology was made, with emphasis on the sinuses. On Oct. 22 an x-ray study of the paranasal sinuses disclosed the frontal sinuses to be well developed and normally aerated. There was no mucosal thickening or bone destruction. Both antra were well developed. There was a slight clouding on the right side as compared with the left, but no evidence of bony destruction and no definite fluid level were noted. There appeared to be some increase in density near the floor of the right antrum, which may have been due to polypoid tissue or excess mucosal thickening. The anterior view showed some delineation near the upper portion, which possibly was due to minimal delineation of ethmoid cellulation. What was present of a sphenoid sinus appeared to be well aerated. The impression was of clouding of the right antrum with the possible presence of a polyp near the floor.

The child still had profuse bilateral rhinorrhea and nasal obstruction and continued to be a mouth breather despite the fact that the tonsils and adenoids had been removed. There was no x-ray evidence to explain the nasal obstruction at this time.

Dr. N. C. Ochsenshirt, an oral surgeon, and Dr. M. S. Kneedler, an otorhinolaryngologist, were called in consultation. After their extensive study, they were inclined to believe that the cause of the nasal respiratory difficulty was obstruction in the choanae rather than pathology in the sinuses.

The family was advised of the seriousness of this type of nasal obstruction, and it was recommended that further consultation be sought.

Three years intervened between removal of the child's tonsils and adenoids and her first visit to me.

Now 8, she was referred to me Oct. 6, 1951, by Dr. Charles Baltimore, of West Mifflin, Pa. Dr. Baltimore's history stated that she was a mouth breather with a chronic prenasal discharge which necessitated constant mopping, but with no postnasal discharge. However, when the child stooped forward, there was an increase in the flow of colorless mucoid nasal secretions.

When I first observed her, she was underweight, pale, and listless and seemed constantly tired. Her parents reported that her sleep had always been disturbed at night (owing to the nasal obstruction) by constant tossing and stertorous raucous breathing, and as a result she would doze off at any time during the day. Her mouth was constantly dry and several times

during the night she would ask for water to moisten her lips. She was unable to blow her nose or draw back any of the secretions into the nasopharynx. There was complete anosmia, and there was no improvement in nasal respiration despite the fact that her tonsils and adenoids had been removed.

My examination (Oct. 6) revealed complete obstruction of both sides of the choanae. I was unable to pass a probe or an infant nasopharyngoscope into the epipharynx through the nose. Further examination revealed a high-arched palate. Aural examination was negative. Pure-tone audiometry and spondee speech tests gave normal results despite the nasal obstruction. The provisional diagnosis was bilateral choanal obstruction, possibly osseous. Arrangements were made to have the patient admitted to Temple University Hospital on my service Oct. 8 for observation and surgery.

Fig. 1.—Sinuses are of good size and well aerated and are normally developed.

Prior to operation roentgenologic studies were done of the paranasal sinuses, nasal passages, and nasopharynx, including high-angle vertex projections and also lateral views with iodized oil (Lipiodol) in the nares. The report stated that examination of the paranasal sinuses disclosed the sinuses to be developed normally. The sinuses were of good size and are well aerated. There was no evidence of polypi, a fluid level, or mucous-membrane thickening in any of the sinuses (Fig. 1).

There was no evidence of a mass in the nasopharynx, and no mass was identified in the region of the choana. The routine anterior projections showed nothing of significance referable to the nasal septum or turbinates, and the right and left nares were aerated. However, lateral views of the nasal region, made with the patient lying supine after instillation of iodized oil into both nares, showed bilateral obstruction in the region of the choanae. Apparently the obstruction was complete to the passage of iodized oil, and none of this radiopaque material reached the nasopharynx (Fig. 2).

Roentgenologic study of the mastoids was also made. The mastoids were found to be equally pneumatized and normal (Fig. 3).

The preoperative diagnosis was complete bilateral osseous obstruction of the choana (congenital).

An operation for removal of the obstruction was done Oct. 19 with endotracheal anesthesia.

Fig. 2.—Patient supine; complete bilateral obstruction in region of choanae, with no iodized oil entering epipharynx.

Fig. 3.—Mastoids equally pneumatized and normal.

The nose was packed with 1:15,000 epinephrine bilaterally. After 10 minutes the packing was removed. The inferior turbinates were infracted upward bilaterally with a Killian speculum. A Doyen Jansen side mouth gage was then inserted. The nose was inspected with the nasopharyngoscope, and both sides of the posterior nares were found to be completely closed. Upon palpation of the epipharynx, the choanae were found to be obstructed. A medium-sized Killian nasal speculum was inserted to aid visualization in the deeper recesses of the nose.

A crisscross incision was made with a long sharp bistoury. Then, with a Freer and Cottle elevator, the mucous membrane of the bony portion of the obstruction was elevated upward, downward, and laterally, thus exposing the bony obstruction. I inserted my left index finger into the mouth and into the epipharynx in order to palpate and guide the movement of a dental bur. With the right hand, a long sharp five-pointed bur attached to a dental engine was inserted into the right side of the nose, and the osseous obstruction was perforated. The remainder of the bone was removed with a biting forceps, and the sharp bony edges were smoothed with the bur, which was constantly guided in the epipharynx by the left index finger. The procedure was repeated on the left side. The crisscross mucous membrane was pushed backward to cover the raw exposed bony surface. To keep the mucous membrane in place and to prevent atresia, a No. 26 French rubber catheter was inserted in the choanae in the following manner:

Fig. 4.—Insertion of No. 26 French catheter in form of rubber "doughnut."

Two soft No. 9 French rubber catheters were passed through the nares into the pharynx to act as pilots. The distal ends of the catheters were grasped with a Kelly hemostat in the oropharynx and were brought out through the mouth. (The technique was essentially the same as for the insertion of a postnasal pack.)

The No. 26 French catheter was then sewed and tied to the proximal ends of the catheters. The catheters were drawn through each side of the nose, the No. 26 French catheter being pulled through the epipharynx, oropharynx, and mouth, the pilot catheters being removed through the mouth. The two loose ends of the No. 26 French catheter were then sewn together so that the catheter was converted into a rubber "doughnut," which remained in the nose. The catheter resembled the ring worn by the Ubangi tribe (Fig. 4).

On Oct. 26, seven days postoperatively, the nasal region was x-rayed for comparison with the preoperative x-rays of Oct. 18. The report showed that the tube was filled with an opaque medium, so that it could be readily visualized. The tube was seen as it passed around the nasal septum posteriorly and through the opposite nares (Figs. 5 and 6).

On the 12th postoperative day, we began to rotate the tube to make sure that there were no synechiae or adhesions. We made three openings in the tubing, one posteriorly in the region of the epipharynx and two in the region of the nasal vestibule. Through these openings in the "doughnut," the child was able for the first time in her life to breathe through her nose.

Fig. 5.—Instilled iodized oil in rubber catheter.

Fig. 6.—Taylor position; rubber tube passing around nasal septum posteriorly and through opposite nares.

She was discharged from the hospital with the "doughnut" remaining in her nose to insure patency of the choanae. The family and the child were instructed to keep rotating the tubing several times daily; these instructions were followed meticulously.

On Nov. 7 the child was readmitted to Temple University Hospital for replacement of the tubing. With general anesthesia, the large tube (No. 26) was removed and was replaced by a smaller (No. 22) French rubber catheter, which also was formed into a "doughnut" (Fig. 7). At this time, both choanae were found to be patent. Three openings were again made in the tube, one posteriorly and two anteriorly, so as to facilitate nasal respiration. The child was discharged from the hospital three days later and again instructed to rotate the rubber tubing.

On Jan. 16, 1952 (48 days postoperatively), she was readmitted to Temple University Hospital for removal of the tubing from the choanae. With narcosis, the "doughnut" was removed, and a Sinexon nasal dilator was inserted through both nostrils. There was no necessity to replace the tubing, since both choanae had remained patent. The patient was discharged from the hospital with instructions to return several times for further observation. She was last seen in November 1952. At that time, with local anesthesia, a complete endonasal examination was made. There was no evidence of mucous-membrane changes due to pressure exerted by the catheters; there was also no evidence of synechiae, and there was perfect nasal respiration.

Fig. 7.—No. 22 French rubber catheter replacing larger (No. 26) catheter in form of rubber "doughnut."

CONCLUSION

Before surgery, the child instinctively breathed through her mouth, but now that the nasal passages were actually open for the first time, she had to reeducate herself and learn that her nose was not there as an ornament but was for breathing purposes.

For the first time in her life she enjoyed the luxury of smelling and tasting food. It appears that the sense of smell is dependent upon air currents to carry the various odors to the olfactory nerve. It was obvious in this case that as soon as aeration was established the child began to detect odors. At first she was bewildered, but she soon learned by experience how to differentiate one smell from another. Actually, she went on a smelling "binge," sniffing one perfume bottle after another. She had often heard remarks made by other children about the offensive odor of a skunk, but she had never actually experienced the sensation. One day while driving around the countryside with her parents, she at long last got a whiff of skunk.

The newly developed sense of smell resulted in an improvement in her sense of taste, and her appetite increased to such an extent that she gained 25 lb. (11.3 kg.) within the year.

COMMENT

The surgical procedure was simple. It was limited to removal of the bony obstruction in the choanal region and is most practical in cases of bilateral atresia. The operation was nondestructive and effective. Patency of the newly created openings was established.

When asphyxia neonatorum is noted, not only should pyloric stenosis, cardiac disease, and thymic enlargement be considered, but also the possibility that a nasal and/or choanal obstruction is present.

I might suggest that when an adenoidectomy is performed, some form of a nasal sound (preferably a Sinexon nasal dilator) be employed to ascertain patency. In routine tonsillectomies and adenoidectomies, I have discovered five unilateral choanal obstructions, of which two were fibrous and three were osseous, because I used the Sinexon nasal dilator; otherwise, I might have overlooked them.

It is interesting to note that, despite bilateral bony obstruction in the region of the choana, the sinuses were fully developed. This fact refutes the long-accepted concept that air pressure plays an important role in development of the sinuses. The patient's sinuses were completely normal, despite the fact that no inspired air had been circulated in them by nasal respiration.

It is noteworthy that the child's hearing was not affected at any time by the obstruction. She always had normal tympanic membranes and patent Eustachian tubes. X-ray examination revealed the mastoids to be pneumatized and normal. Obviously, nature compensated for the obstruction so far as her hearing was concerned. Normally, air reaches the Eustachian tube through the anterior nares and mouth. In this case air was inspired solely through the mouth. The child learned to regulate her middle-ear and Eustachian-tube air pressure by deglutition. Apparently, her regulatory mechanisms were functioning in such a manner that she managed to get a normal air flow to fill the middle ear, and, on the other hand, she never overloaded the tympanic cavity; otherwise, the middle ear would have shown some damage.

This case demonstrates that the sense of smell is dependent upon air currents to carry odors to the olfactory nerve. The anosmia was caused by choanal obstruction because of faulty aeration.

Last, but not least, normal physiologic nasal respiration was established.

PINEALOCYTOMA WITH MENINGITIS

MIHAJLO PRAZIC, M.D.
AND
NENAD GRCEVIC, M.D.
ZAGREB, YUGOSLAVIA

THE EPIPHYSIS, or pineal gland, is not usually encountered in otolaryngology. The case which we have observed seems therefore worth noting.

About the pineal gland nothing certain is as yet known. It has not been determined whether or not it has an endocrine function.[1] Experimental results [2] with implantation, extirpation, and injection of pineal gland tissue in animals are so contradictory that they do not allow definite conclusions. Many authors [3] are of the opinion that the pineal gland is a vestigial organ of the parietal eye.

Tumors of the pineal gland are relatively rare. Bailey and Cushing [4] found that among tumors of the brain only 0.7% were pineal tumors. In most of the published cases, moreover, the tumors have been found only at autopsy. The Institute of Pathology of Zagreb has found only four pineal tumors during the last 25 years, and only one of them was diagnosed during life. This late diagnosis is due to the vague clinical picture which such tumors yield and to the location of the pineal gland, which is situated in the recessus pinealis of the third ventricle above the corpora quadrigemina.

Pineal tumors seem to grow slowly. They appear mostly in young people before puberty, more often in boys than in girls.[5] They vary considerably in size. Sometimes a tumor only a little larger than a normal pineal gland gives rise to symptoms, but more frequently a larger tumor gives only vague symptoms and in rare cases none at all.

The symptoms which may be caused by pineal tumors can be grouped as follows:

1. Symptoms of higher intracranial pressure resulting from compression of the aquaeductus Silvii. This compression may be a complete one, which is extremely

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Read at the meeting of the Ear, Nose, and Throat Section of the Medical Society of Croatia, Sept. 17, 1952.

From the Department of Otolaryngology (Dr. Gušić, Chief), and Institute of Pathology (Dr. Saltykow, Chief), Medical Faculty, University of Zagreb.

1. Grollman, A.: Essentials of Endocrinology, Ed. 2, Philadelphia, J. B. Lippincott Company, 1947. Bargmann, W.: Die Epiphysis cerebri, in Handbuch der mikroskopischen Anatomie des Menschen, edited by W. Möllendorff, Berlin, Springer-Verlag, 1943. de Gennes, L.: Maladies des glandes endocrines, Paris, Ernest Flammarion, 1949.

2. Globus, J. H.: Pinealoma, Arch. Path. **31**:533, 1941.

3. Gladstone, R. J., and Wakeley, C. P. G.: The Pineal Organ, Baltimore, William Wood & Company, 1940.

4. Bailey, P., and Cushing, H. W.: Die Gewebs-Verschiedenheit der Hirngliome und ihre Bedeutung für die Prognose, Jena, Germany, Gustav Fischer, 1930.

5. Zülch, K. J.: Die Hirngeschwülste, Leipzig, J. A. Barth, 1951.

rare, or a partial one, which is more common. The partial compression leads to hydrocephalus. High intracranial pressure is accompanied by such typical symptoms as headache, vomiting, nausea, papillary stasis, and somnolence.

2. Symptoms arising from the pressure on the corpora quadrigemina and adjacent structures. This pressure may cause oculomotor troubles such as impossibility of looking upwards, anisocoria, strabismus, diplopia, and medial rectus paralysis. A rigidity of the pupils is manifested in 50% of all cases. The incidence of all other symptoms is only 30%.[6] Deafness of the central type occurs when the pressure reaches the lateral lemniscus.

3. Cerebellar symptoms arising from the pressure under the tentorium. These symptoms include nystagmus, ataxia, asynergia, and adiadochokinesia. In the case of a compression of the cerebral peduncle, spastic paralysis of the limbs and paralysis of the sixth nerve can be found.

4. Endocrine troubles. Pubertas praecox, dystrophia adiposogenitalis, hypogonadism, diabetes insipidus, and obesity occur very seldom.[7] However, many authors are of the opinion that most of the endocrine troubles occur as a result of the pressure on the vegetative centers of the midbrain.[8]

Thus, the symptomatology of pineal tumors is manifold and uncertain, and therefore the majority of all cases remain unrecognized.

The treatment of pineal tumors was at one time surgical, but results were so bad that now only temporal decompression with x-ray treatment is advised.

REPORT OF A CASE

A boy aged 17 years gave the following anamnestic data. His mother had died recently of tuberculosis of the lung. His father and his brothers and sisters were in good health. He had never before been seriously ill and did not remember any childhood diseases. He had always been in good physical condition and at the time of admission was employed as a qualified worker. After a cold a year before, his left ear had begun to discharge. Later it gave him no trouble, and he noticed only a reduction of hearing on the same side. Four months before admission, i. e., immediately after the death of his mother, he had begun to feel ill, had lost his appetite and perspired during the night, but he had continued to work. He went to see a physician and received some relief. Two weeks before his admission there was a sudden onset of high fever, headache, and vomiting. Two days later his neck became stiff, and he went to see a physician, who, suspecting tuberculous meningitis, sent him to the Hospital for Infectious Diseases. There the patient was found to be in good general condition, conscious, and without fever.

Photophobia was present. The lungs showed a specific process in the left upper lobe. The pupils were narrow and equal, and reacted to light and in accommodation. All tested reflexes were present and normal. The urine showed nothing abnormal. The blood count showed 7,800 leucocytes; other clinical and laboratory examinations showed no pathological change. Spinal puncture revealed that the cerebrospinal fluid was slightly opaque, and the pressure increased. Pandy and Nonne-Apelt reactions were positive. Cells numbered 100/3; most were mononuclear. Sugar content was 69 mg. per 100 cc., and albumin, 70 mg. per 100 cc. Because tuberculous meningitis was first suspected, streptomycin was administered. The following day the clinical picture remained the same.

The otologist found the patient quiet and conscious. A meningitis syndrome was clearly present. There was no diplopia. The function of the eye muscles was normal. Nystagmus was

6. Posner, M., and Horrax, G.: Eye Signs in Pineal Tumors, J. Neurosurg. **3**:15, 1946.

7. Russell, W. O., and Sachs, E.: Pinealoma: Clinicopathalogic Study of 7 Cases with Review of Literature, Arch. Path. **35**:869, 1943.

8. Globus.[2] Gladstone and Wakeley.[3]

present. From the left ear there was severe discharge. Examination showed total defect of the tympanic membrane and the lateral wall of the attic. In the middle ear cavity there were purulent cholesteatomatous masses. The fistula symptom was present. Examination with tuning forks showed considerable impairment of the hearing in the left ear.

The patient was immediately transferred to the Ear, Nose, and Throat Clinic, and a radical mastoidectomy was performed. A cholesteatoma as large as a cherry was found. In the area of the tegmen tympani the dura was covered with yellowish layers. On the lateral semicircular canal there was a fistula. Spinal puncture gave a clear fluid with a positive Pandy reaction; the cells numbered 150/3 and were mostly neutrophiles. After the operation we began intralumbar injections of penicillin. During the days following the operation, the patient felt quite well, and his temperature was normal. In the cerebrospinal fluid the cell count increased to 1090/3 and consisted mostly of neutrophiles. At each lumbar puncture the fluid was under increased pressure, which would become normal after the removal of 20 to 30 cc. of fluid. The patient did not complain about headache and had an excellent appetite.

Fig. 1.—Tumor of the pineal gland protruding between the cerebrum and the cerebellum and lying above the tentorium.

On the seventh day following the operation, the patient became a little excited, and there was a slight hyperesthesia of the skin. The temperature was normal. The operative cavity looked clean and fresh. There was no nystagmus. The cerebrospinal fluid was clear, and the hypertension was less marked. The lymphocytes numbered 36/3. On the following day, the patient was in a state of excitation and hyperesthesia. The temperature was normal. The spinal puncture showed a clear and hypotensive fluid. The patient complained of severe headache. That night he suddenly died.

Autopsy showed the following: The brain was swollen on the surface and smooth; the sulci and gyri were flattened. The dura and pia were smooth and shiny. The arteries at the base of the brain were without pathologic changes. The inner surface of the dura was without changes. At the lamina quadrigemina (Fig. 1), on the site of the pineal gland, there was a tumor as large as an egg; it was soft, dark red, and uneven. This tumor was exerting pressure against the aquaeductus Silvii; it protruded between the cerebrum and the cerebellum, and lay above the tentorium. The lateral ventricles and the third ventricle were enlarged and filled with clear liquid. The fourth ventricle was normal, the ependyma, smooth. In the upper lobe of the left lung there was a tuberculous node about as large as a walnut with a cavity in it. The external and internal genital organs and all endocrine glands were without change.

Microscopically, this tumor of the pineal region consisted of large spheroidal cells with large nuclei (Fig. 2). These cells had an alveolar arrangement. Among them there were some small cells resembling lymphocytes. In the tissue were many blood vessels, and in many places there was evidence of hemorrhage and necrosis (Fig. 3).

The pathological diagnosis was pinealocytoma, internal hydrocephalus, cerebral edema, and tuberculous cavity of the apex of the left lung.

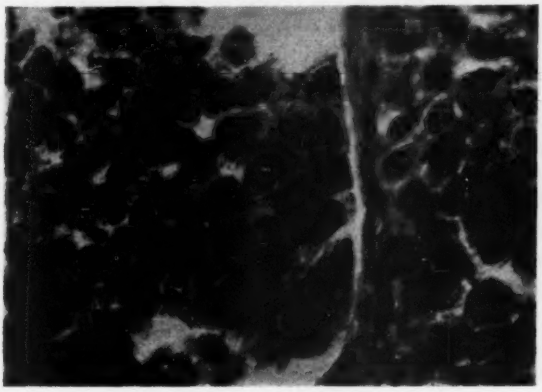

Fig. 2.—Photomicrograph of pineal tumor showing large spheroid cells with large nuclei in alveolar arrangement (× 300).

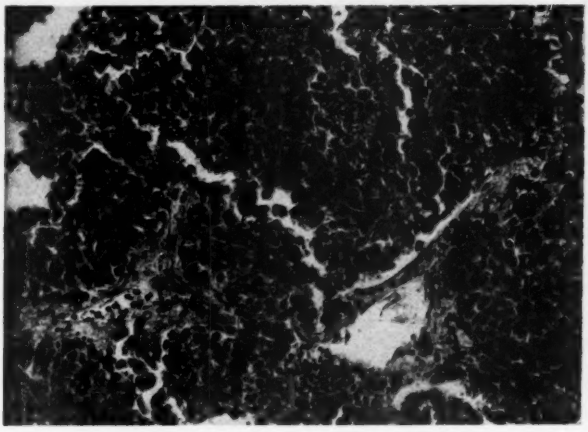

Fig. 3.—Photomicrograph of pineal tumor tissue showing blood vessels and evidence of hemorrhage and necrosis (× 50).

COMMENT

In this case, a large tumor of the pineal gland which did not give any symptoms caused death. The patient also suffered from otitis media with cholesteatoma and later from meningitis. After the operation the patient felt quite well, but on the eighth day after the operation he suddenly died. The autopsy showed that two different conditions were present, of which only the tumor of the pineal gland

could be seen, the cholesteatomatous otitis and meningitis having been cured. Trauma from surgical intervention was so severe that it resulted in multiple hemorrhages inside the tumor. These hemorrhages led to a gradual enlargement of the tumor. Eventually this enlargement caused a complete compression of the aquaeductus Silvii and of the centers of the mesencephalon, with consequent sudden death. Because the tumor had the tendency of growing toward the posterior fossa and above the tentorium it manifested none of the above symptoms so long as it was not too large. Owing to hemorrhage it became enlarged and caused sudden death.

SUMMARY

A case of tumor of the pineal gland with otitis media and meningitis is reported. The symptomatology of pineal tumors is uncertain and not easily distinguishable. In this case there were no symptoms. The patient underwent operation for cholesteatomatous otitis and meningitis, but after initial recovery he died suddenly, because of hemorrhage in the tumor as a consequence of surgical trauma. The hemorrhage caused the tumor to enlarge in such a way that death resulted from pressure on the centers of the mesencephalon.

Special Reports

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

AS OF JULY 1, 1953

Audicon Models 400, 415, 530, & 615

Mfr., National Earphone Co., Inc.
20 Shipman St.
Newark 2, N. J.

Audiotone Model 11

Mfr., Audio Company of America
5305 N. Sixth St.
Phoenix, Ariz.

Audivox Models Super 67 & 70

Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Aurex Models L & M

Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Beltone Mono-Pac Model "Lyric"

Beltone Mono-Pac Model M

Beltone Mono-Pac Model "Rhapsody"

Beltone Symphonette Model

Mfr., Beltone Hearing Aid Co.
2900 W. 36th St.
Chicago 32

Cleartone Models 500 & 700

Cleartone Regency Model

Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Dahlberg Model D-1

Dahlberg Junior Model D-2

Dahlberg Model D-3 (Tru-Sonic)

Dahlberg Model D-4 (Tru-Sonic)

Mfr., The Dahlberg Company
Golden Valley
Minneapolis 22

Dysonic Model 1

Mfr., Dynamic Hearing Aids, Inc.
149 Church St.
New York 7

Fortiphone Models 19LR, 20A, 21C, & 22

Mfr., Fortiphone Limited
Fortiphone House
247 Regent St.
London, W.1, England

Distr., Anton Heilman
75 Madison Ave.
New York 16

Gem Models V-35 & V-60

Mfr., The Gem Ear Phone Co., Inc.
50 W. 29th St.
New York 1

Goldentone Models 25, 69, & 97

Mfr., Johnston Hearing Aid Mfg. Co.
708 W. 40th St.
Minneapolis 8

Distr., Goldentone Corporation
708 W. 40th St.
Minneapolis 8

Maico Model J

Maico Quiet Ear Models G & H

Maico Top Secret Model L

Maico UE Atomeer

Mfr., The Maico Company, Inc.
21 N. 3rd St.
Minneapolis 1

Mears (Crystal & Magnetic) Aurophone Model 200

Mfr., Mears Radio Hearing Device Corporation
1 W. 34th St.
New York 1

Micronic Model 303

Micronic Model Mercury

Micronic Star Model

Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Microtone Classic Model T-9

Microtone Models T-10, T-612, & 45
Mfr., The Microtone Company
Ford Parkway on the Mississippi
St. Paul 1

National Cub Model (C)

National Model D (Duplex)

National Standard Model (T)

National Star Model (S)

National Ultrathin Model 504

National Vanity Model 506
Mfr., National Hearing Aid Laboratories
106 S. 7th St.
Philadelphia 6

Normatone Model C
Mfr., Johnston Hearing Aid Mfg. Co.
708 W. 40th St.
Minneapolis 8
Distr., Normatone Hearing Aid Co.
22 E. 7th St.
St. Paul 1

Otarion Model E-4

Otarion Models F-1, F-2, & F-3

Otarion Models G-2 & G-3

Otarion Model H-1
Mfr., Otarion, Inc.
4757 N. Ravenswood Ave.
Chicago 40

Paravox Model D (Top-Twin-Tone)

Paravox Model J (Tiny-Myte)

Paravox Model Y (YM, YC, & YC-7) (Veri-Small)
Mfr., Paravox, Inc.
2056 E. 4th St.
Cleveland 15

Radioear All-Magnetic Model 55

Radioear Permo-Magnetic (Multipower)

Radioear Model 62 Starlet

Radioear Model 72

Radioear Model 82 (Zephyr)
Mfr., E. A. Myers & Sons, Inc.
306 Beverly Rd.
Mount Lebanon
Pittsburgh 16
Distr., Radioear Corporation

Silvertone Models J-92 & P-15
Mfr., Johnston Hearing Aid Mfg. Company
708 W. 40th St.
Minneapolis 8

Solo-Pak Model 99
Mfr., Solo-Pak Electronics Corp.
Linden St.
Reading, Mass.

Sonotone Models 900, 910, 920, 925, 940, & 966
Mfr., Sonotone Corporation
Elmsford, N. Y.

Super-Fonic Hearing Aid
Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Televox Model E
Mfr., Televox Mfg. Company
1307 Sansom St.
Philadelphia 7

Telex Models 97, 99, 200, 300-B, 400, 500, 952, 953, & 1700
Mfr., Telex, Inc.
Telex Park
St. Paul 1

Tonamic Model 50
Mfr., Tonamic, Inc.
12 Russell St.
Everett 49, Mass.

Tonemaster Cameo Model

Tonemaster Model Royal
Mfr., Tonemaster, Inc.
400 S. Washington St.
Peoria 2, Ill.

Unex Midget Models 95 & 110

Unex Models 200 & 230
Mfr., Nichols & Clark
Hathorne, Mass.

Vacolite Models J & J-2
Mfr., Vacolite Company
3003 N. Henderson St.
Dallas 6, Texas

Zenith Miniature 75
Zenith Model Royal
Zenith Model Super-Royal
Zenith "Regent"
Mfr., Zenith Radio Corporation
5801 W. Dickens Ave.
Chicago 39

(All the accepted hearing devices have vacuum tubes.)

Accepted hearing aids more than five years old have been omitted from this list for brevity.

SEMIPORTABLE HEARING AIDS

Ambco Hearing Amplifier (Table Model)
Mfr., A. N. Brooks Company
1222 W. Washington Blvd.
Los Angeles 7

Aurex Semi-Portable
Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Precision Table Hearing Aid
Mfr., Precision Hearing Aids
5157 W. Grand Ave.
Chicago 39

Sonotone Professional Table Set Model 50
Mfr., Sonotone Corporation
Elmsford, N. Y.

Abstracts from Current Literature

Ear

MASTOIDITIS IN THE INFANT: CONCEPT AND DIAGNOSIS. R. RAMOS and R. PACH VINALS, Arch. pediat. **2**:219 (Sept.) 1950.

Acute mastoiditis is still far too common in Spain. The authors believe the reason to be that the diagnosis is sometimes difficult for the general practitioner to make. They describe the anatomy of the mastoid antrum in the infant and the ease with which it may become involved during an otitis media infection. This may occur without much evidence of drainage from the ears.

The diagnostic signs, both clinical and roentgenologic, are discussed, with a number of illustrations. General symptoms and those referable solely to ears are discussed, as well as the types of illnesses in which mastoiditis may be a concomitant or sequel; however, the author says it must always be remembered that it is an infection and not just an illness which coexists with the nutritional and diarrheal diseases of infancy.

The techniques of transillumination and the procedure for the drainage of the mastoid are given. If antibiotics do not give immediate relief, one should use x-rays and possibly operate for drainage.

Mastoiditis due to tuberculosis is nearly always fatal because it is usually found along with generalized tuberculosis or with meningitis, and unless diagnosed very early it is very unlikely to be amenable to treatment.

MARY ALLEN, San Francisco [A. M. A. Am. J. Dis. Child.].

THE STATE OF ILLINOIS COOPERATES WITH THE OPHTHALMOLOGIST AND OTOLARYNGOLOGIST. HERBERT R. KOBES, Illinois M. J. **99**:303 (June) 1951.

Kobes points out that many children in Illinois with hearing difficulties have needed help through medical facilities. Private physicians, school health programs, the hospitals, the Division of Services for Crippled Children, the Department of Public Welfare through the School for the Deaf, the Division of Education for Exceptional Children, and the Division of Vocational Rehabilitation give through their combined resources programs which are probably not excelled.

Since its inception the Illinois program for the child with hearing loss has been developed from the "Syllabus on Hearing Conservation" of the Academy of Ophthalmology and Otolaryngology. From the family physician, often through the health department, the cases are referred to the Division of Services for Crippled Children for further evaluation and planning for future care.

The Division's clinics are held in about 40 different communities throughout the state. Here the child is seen by a pediatrician and a speech and hearing consultant. If a patient is found to have a loss of 20 or more decibels in two or more frequencies in the same ear, the patient is referred to an otologist certified by the American Board of Otolaryngology for definitive medical diagnosis and plan of treatment at the clinic; a public health nurse or medical social worker may aid in interpreting to the family what may need to be done for the child. The otologist reports his findings to the division. Office treatments, hospital care for surgery, hearing aids and training in their use, school seating placements, and lip reading instruction are among the recommendations that are made. Through the resources of the Division for Crippled Children all children at the School for the Deaf who should have hearing aids to develop their educational

program to the fullest extent are able to obtain aids. This has resulted in graduating a considerable number of children from this residential school to schools in their own home community—even the regular classrooms.

Efforts are directed toward educating physicians, public health workers, and the lay public to the need of making the earliest possible diagnosis for children born with a severe hearing loss or deafness. Under this educational program during the past five years more and more preschool deaf children are found annually, and the long tedious program of training and education is begun for the children and their parents. Mothers and their children aged from 2 to 5 years come to Jacksonville for 10 days to the annual Institute for Mothers of Pre-School Deaf Children. Lectures by authorities in child care and the medical and education problems of the deaf are combined with demonstrations concerning the training that needs to be followed to gain speech for many of these children.

This program has been carried out for four years. It has become evident that there is need for medically trained men with training in lip or speech reading, in acoustical training, and in hearing-aid fitting. Courses teaching the elements of complete rehabilitation in this field are available on a postgraduate level. During the past summer some 30 public health nurses had an intensive two-week course in hearing testing to fit them into community conservation of hearing programs. Courses are offered at various universities in the training of teachers of the deaf and hard of hearing. In the educational institutions concerned with training of handicapped children, one frequently finds severe social and educational impairment which becomes relieved when a further diagnostic evaluation reveals a second disorder and thus makes possible a different educational program for the child. The visually handicapped child who does poorly in the sight-saving class may have a serious hearing loss, too.

BARBOUR, Peoria, Ill. [A. M. A. Am. J. Dis. Child.].

AN UNUSUAL CASE OF UNILATERAL EIGHTH NERVE TUMOR. V. H. MARK and W. H. SWEET, J. Neurosurg. **9**:395 (July) 1952.

The occurrence in the younger age group of bilateral acoustic tumor, or acoustic tumor associated with Recklinghausen disease, or neurofibromatosis, is well known. Cushing stated that "a patient under 20 with a cerebellopontile-angle syndrome in all likelihood has a lesion other than an acoustic tumor."

The case of unilateral acoustic nerve tumor presented by the authors proves to be an exception to that rule, for the age of onset was 6 years and the neuroma was removed and the diagnosis verified histologically when the boy was 8½ years old. Furthermore, the patient showed none of the associated stigmas of Recklinghausen disease, as determined either by physical examination or from the family history. Other unusual features of this case include normal spinal fluid protein and massive destruction of the pyramid of the involved petrous bone.

ALPERS, Philadelphia [A. M. A. Arch. Neurol. & Psychiat.].

ON THE PHENOMENON OF PARACUSIS WILLISI. L. HALPERN, Harefuah **43**:123 (Nov.) 1952.

In 1672 Willis observed in his book "De anima brutorum" that deaf people sometimes hear better in the presence of noise. There has as yet been no satisfactory explanation of this phenomenon. Hearing and vibration resemble each other and seem to be functionally related. They both pick and perceive rhythmic oscillation. It is also noted that the fish keeps in contact with its environment by perceiving vibrations in the water. Deaf people can be aware of passing vehicles or the noise of a pounding hammer due to transmitted vibratory sensation. Vibration apparently has an effect on the auditory threshold and improves hearing ability. Usually hearing and vibration together facilitate perception of the outer world.

SAVITSKY, New York [A. M. A. Arch. Neurol. & Psychiat.].

SPHYGMO-AUDIOMETRY: THE NEW METHOD TO TEST OBJECTIVE HEARING. A. VASSALLO DE MUMBERT, Bol. españ. otorrhinolaring. y bronchoesofagol. **5**:37, 1952.

De Mumbert describes a new technique for testing objective hearing. This technique, which he has developed accidentally, occurred to him while examining a patient. He coordinates the radial pulse pressure with the various frequencies of the tonal scale and notes the definite changes

in the pulse pressure with the increase in intensities at the various frequency levels. This is based on the fact that the acoustic and the vestibular triangular nuclei situated in the floor of the fourth ventricle have an intimate relationship through a somaticovegetative anastomosis with the nucleus of the vagus nerve. Hence, the stimulus to the acoustic nuclei would cause a marked vasodepressor action on the circulatory system, thus causing a momentary fall in arterial pressure. After making these observations de Mumbert constructed a suitable apparatus for recording automatically on a dial the blood pressure reading and the influences on the curve by variations in the intensity at the definite frequencies. This method of examination is extremely important because it can be used in testing very small children, even infants; older children who do not cooperate properly with other procedures; adults whose objective responses are obtained without any volnutary influence on the part of the patients; deaf-mutes; mentally retarded patients; patients with psychogenic disturbances, and those patients presenting medicolegal complications. This method is well worth investigating.

PERSKY, Philadelphia.

Pharynx

RHYTHMIC PALATOPHARYNGEAL MYOCLONUS. MORTON M. STERN, J. Nerv. & Ment. Dis. **1**:48 (Jan.) 1949.

Stern reviews the syndrome of rhythmic palatopharyngeal myoclonus and reports a case in which it was associated with severe closed head trauma. The patient had ben unconscious for several hours; on the 21st day he noticed constant clicking noises, especially in his left ear. Examination of the pharynx revealed rhythmic, synchronous bilateral myoclonic movements of the soft palate, uvula, pharynx, and base of the tongue, at a rate of 120 to 140 per minute. When the mouth was open, there were periods of 5 to 10 seconds in which the movements were barely perceptible; swallowing and coughing decreased this latent period. The movements were synchronous with the clicking noises, and the latter persisted during sleep. Pressure on the eyeballs or the carotid arteries caused a momentary increase in the rate. Fluoroscopic examinations revealed no myoclonic movements of the diaphragm, lungs, stomach, or esophagus. Over a period of four years there has been no response to any medication or change in the clicking noises, but the palatal myclonus is harder to elicit.

The author believes that the syndrome in this case could be due to thrombosis of a small branch of the left vertebral artery affecting the olivodentate-tegmental tract complex.

The author compares the syndrome to the rhythmic reciprocating expansion and contraction of the oral cavity with corresponding relative negative and positive pressures, such as in gill-breathing fishes. He points out (1) that the evolution of lungs in the higher vertebrates dispensed with the branchial structure's function of water breathing and that in humans the first branchial arch and the first branchial pouch furnish the very structures that are involved in rhythmic palatal myoclonus, and (2) that the olivodentate complex is poorly differentiated in fishes but maximally developed in man. Thus, the author considers rhythmic palatopharyngeal myclonus as an accessory respiratory reflex that becomes active when its regulators are damaged.

FARMER, Philadelphia [A. M. A. Arch. Neurol. & Psychiat.].

THE SURGICAL TREATMENT OF CANCER OF THE TONGUE. WILLIAM J. MACFEE, New York J. Med. **53**:163 (Jan. 15) 1953.

Carcinoma of the tongue has long been considered a disease of males, the percentage being from 80 to 90, in contrast to an average percentage of 10 to 20 in females. However, it has been said that cancer of the tongue is beginning to increase among women. Nobody yet knows the cause of cancer, but tobacco, ill-fitting dentures, carious teeth, and excessive use of alcohol are thought to be irritating agents which may superinduce cancer. Reports from South India, where the natives hold a quid of tobacco, lime, and betel leaf in the mouth, show frequent cancer incidence. Syphilis, diabetes, glossitis, and leucoplakia may well be prodromal to cancer. Vitamin deficiency, especially of B-complex, is responsible for many cancerous conditions. Deficiencies of minerals, such as iron and copper, are noteworthy. Dentists are now doing well

in suspecting cancer in the mouth and are referring patients to their physicians. Manifestations are limitations in protrusion of the tongue, stiffness, speech awkwardness, excessive salivation, and absence of mercurial treatment, all of which are to be recorded in a patient's history. Tongue appearance is important, e. g., "frosty tongue," induration on palpation, leucoplakia, or other abnormalities. Since few physicians examine the base of the tongue and larynx with a mirror, cancer may obtain a good start before being recognized. The anterior two-thirds of the tongue, the posterior third of the dorsum, the inferior basal region, and the tongue tip, in that order, are the common loci. Treatment which was formerly largely surgical has been replaced partially by radiation. At present surgery is preferred by many physicians, although the implantation of radium seeds is still being done. Apart from all other considerations, early diagnosis is paramount.

I. W. VOORHEES, New York.

ACCELERATION OF DEVELOPMENT AND HYPERTROPHY OF THE TONSILS: CONTRIBUTION TO THE BIOLOGY OF THE TONSILS ACCORDING TO AGE. BERNFRIED LEIBER, Med. Klin. **47**:582 (April 25) 1952.

Like other phases of development, hypertrophy of the tonsils has accelerated within recent years. For instance, in 1925 only 0.2% of all cases of hypertrophy of the tonsils were found during the first year of life, while in 1950 the percentage was 3.6. The maximal percentage is reached at an earlier age, and the involution also occurs at an earlier age. Greater details are given in other publications.

AMBERG, Rochester, Minn. [A. M. A. Am. J. Dis. Child.].

Larynx

FOREIGN BODIES, EXOGENOUS AND ENDOGENOUS, IN THE RESPIRATORY TRACT. R. RAMOS and R. PACH VINALS, Arch. Pediat. **11**:557 (March-April) 1952.

Eleven cases with x-rays and complete clinical histories of foreign bodies in various parts of the respiratory tract are presented. Some cases were emergencies and needed immediate attention, while others, because of their location, had been incorrectly diagnosed. A lung abscess developed in a child following aspiration of a small portion of a peanut which lodged in a small bronchiole. The child had been treated at intervals for various upper respiratory infections for a total of 15 months, and only when the vomiting and odor suggested an abscess was a true diagnosis made.

Another patient had been treated for pertussis and bronchopneumonia, but the true cause was not determined for a year. Various other cases in which foreign bodies of vegetable origin were involved—peanuts, beans, etc.—and metallic objects, such as coins and buttons, are reported. Endogenous foreign bodies due to laryngotracheobronchitis, necrotic tracheobronchitis, endotracheal abscesses, diphtheria, tuberculosis of tracheobronchial lymph nodes, and neoplasms are also discussed. Atelectasis may result from any one of these diseases, and in the case of a 5-year-old girl with a rupture of a tuberculous cavity into the bronchus this occurred. The caseous material acted as a foreign body. Besides the surgical or bronchoscopic intervention, antibiotics are always indicated. In the case of the child with tuberculosis, streptomycin was given before bronchoscopy.

MARY ALLEN, San Francisco [A. M. A. Am. J. Dis. Child.].

HEMANGIOMA OF THE LARYNX IN AN INFANT. HAROLD W. DARGEON, and JOHN F. DALY, J. Pediat. **39**:738 (Dec.) 1951.

A 3-month-old infant had progressive and eventually fatal respiratory distress after a prophylactic inoculation of diphtheria, pertussis, and tetanus vaccine. The child had a capillary hemangioma of the larynx, the 11th such reported case in the literature, probably not a true index of the frequency of this condition.

MACDONALD, Pittsburgh [A. M. A. Am. J. Dis. Child.].

Nose

TRIGEMINAL NEURALGIA. M. M. PEET and R. C. SCHNEIDER, J. Neurosurg. **9**:367 (July) 1952.

The authors report the data on 689 patients with trigeminal neuralgia who were examined over a 10-year period. The data concern age and sex incidence, side of involvement, duration of symptoms, division involved, trigger zones, preoperative complications, operations, immediate complications, postoperative problems, and number of deaths. Follow-up study was accomplished by a questionnaire, to which 65% of the patients replied.

The series comprised 553 patients who had trigeminal rhizotomy, 49 who had received only alcohol injections, and 87 others for whom the diagnosis was made but who were not treated.

From this survey, Peet and Schneider concluded that trigeminal rhizotomy is a much more satisfactory procedure than alcohol injection in the average case of trigeminal neuralgia, for there was complete relief of tic pain in all but 5.4% of the patients treated surgically. Alcohol injection in this series relieved only 15.2% of the patients for longer than one year.

Unpleasant postoperative symptoms, such as crawling and burning paresthesias, occurred in a fairly high percentage of the rhizotomy patients, approximating the incidence of these symptoms after alcohol injection. However, the great majority of patients are so relieved because of the cessation of their severe tic pain that they find these discomforts endurable. The authors believe that proper forewarning of the patients about such possible postoperative symptoms is important and rewarding.

The operative mortality rate in the entire series was 1.6%. The procedures were carried out by 14 surgeons, and 34% of the patients were over 65.

In only 6.5% of the patients who underwent operation did facial paralysis occur, and in no known instance in this series was it permanent.

ALPERS Philadelphia [A. M. A. Arch. Neurol. & Psychiat.].

Miscellaneous

TONSILLECTOMY AND POLIOMYELITIS: II. FREQUENCY OF BULBAR PARALYSIS, 1944-1949. MORRIS SIEGEL, MORRIS GREENBERG, and CATHERINE MAGEE, J. Pediat. **38**:548 (April) 1951.

Since 1910, when a possible relationship between bulbar poliomyelitis and tonsillectomy was first reported, medical opinion on this subject has been divided. The pediatricians support the theory, while the throat surgeons oppose it. Here, Drs. Siegel, Greenberg, and Magee did add fuel to the pediatric fire. They studied 194 cases of bulbar poliomyelitis occurring within one year after tonsillectomy. The number of cases is admittedly small, but they found a significantly higher incidence of bulbar paralysis in the group studied one month after operation. They also found an increase in the mortality rate in those cases seen within two months after operation. The percentage of cases was somewhat higher in the spring and fall than in the summer, and it would seem that bulbar paralysis after tonsillectomy is more frequent in nonepidemic than in epidemic years. The winter months are the safest for tonsillectomy, at least from the poliomyelitis point of view. In a five-year period, no post-tonsillectomy bulbar poliomyelitis was seen in New York during the winter months.

MACDONALD, Pittsburgh [A. M. A. Am. J. Dis. Child.].

CORTISONE THERAPY OF PENICILLIN REACTIONS. JEFF DAVIS, New York J. Med. **51**:69 (Jan. 1) 1953.

Davis states that over-all reactions to penicillin have an incidence of 8 to 10%. These affect chiefly the skin and are a source of great discomfort to the patient. Two kinds of reactions are seen: (1) the early type, which takes place within 24 to 48 hours, and (2) the remote type, or so-called "serum sickness," which comes on 5 to 10 days after the penicillin has been given. Treatment heretofore has been discontinuance of use of penicillin, the administration of histamines, of epinephrine subcutaneously, and of calcium gluconate intravenously, each with only a modicum of success. In cortisone one appears to have a specific agent against urticaria,

erythema multiforme, and various other signs of skin intolerance to penicillin. Five cases are reported, comprising one instance each of cancer of the breast, prostatic carcinoma, and multiple furunculosis, and two cases of adenoma of the thyroid. Within five or six hours complete relief was obtained. Recurrence of the skin lesions was not experienced. In no instance did the total dose of cortisone exceed 100 mg. Davis regards this therapy "definite and safe."

ABSTRACTER'S COMMENT: It seems that in all these patients except the one with furunculosis, penicillin was given postoperatively as a preventative of complications. One would like to know whether the dermal reactions are as likely to occur in the presence of infections associated with marked fever. Further elucidation will be helpful when other experiences with untoward reactions to penicillin are fully recorded.

I. W. VOORHEES, New York.

RECURRENT HEADACHES IN CHILDREN: A STUDY OF 100 CLINIC CASES. GEORGE R. KRUPP and ARNOLD P. FRIEDMAN, New York J. Med. **53**:43 (Jan. 1) 1953.

This is a report on 100 children observed at the Montefiore Hospital, of whom 75 had migraine and 17 had tension headaches. A diagnosis of migraine is made when the pain is unilateral and is thought to be due to vasoconstriction of the cerebral arteries, especially of the branches of the unilateral external carotid. This phase is followed by distention and increased amplitude of pulsation. Tension headaches are often accompanied by emotional conflicts—anger, worry, and frustration. They are bilateral, frontal, or occipital. Frequency and duration are variable, but the headache "may last for weeks." Before these cases were studied a history of the patient and of his parents was recorded, and a thorough physical examination, included investigation of neurologic factors, electroencephalography, studies of visual fields, and blood studies. Psychologic tests were given at times. It was found that most migraine headaches appeared before the patients were 4 years old, difference in sex was of no importance, and 85% of the patients observed suffered from headaches. Children showed prodromal signs of gastric, visual, and psychologic disturbances. Arguments with their parents sometimes produced headaches in both parents and children! The child with a high I. Q. seemed to be more subject to head pains. Medical treatment was with acetylsalicylic acid or 1 mg. of ergotamine tartrate plus 100 mg. of caffeine (Cafergot). The maximum dose (four tablets) was given at intervals of a half-hour to one hour. Improvement sometimes was found after psychiatric investigation; this fact tends to prove the psychosomatic element. Adults should learn to observe a child's moods and not to enter into conflicts at an inopportune time. The prognosis for children is generally poor, since most adults with migraine were sufferers from headaches in childhood (at least 35% of them).

I. W. VOORHEES, New York.

PRACTICAL ASPECTS OF THYROIDECTOMY. BERNARD J. FICARRA, New York J. Med. **53**:215 (Jan. 15) 1953.

Ficarra reviews the surgical anatomy of the region concerned and describes the technical procedure used by him. The laryngologist is concerned with the laryngeal nerves both before and after thyroid surgery. It is well to remember that the inferior laryngeal nerve is composed of two bundles of nerves—one for the separator and the other for the approximator muscles. The lesion most frequently seen postoperatively is paralysis of the recurrent laryngeal nerve on the left with the cord in the so-called "cadaveric position." In cases in which the inferior laryngeal nerve is severed or impinged upon on both sides, the cords are set in approximation, making breathing difficult and giving the voice a crowing quality. The cords cannot be separated. This is so-called abductor or separator paralysis. Tracheotomy is necessary if breathing becomes too difficult.

Nerve identification at operation would tend to avoid accidental cutting of the nerve supply to these muscles.

Postoperative care calls for an intravenous injection of 5% dextrose in water or in saline solution, depending upon the age of the patient and his salt status—loss of salt through excessive perspiration. Fifteen grains (0.97 gm.) of sodium iodide is given with the dextrose. An ice collar is kept on the neck for 24 hours in order to lessen edema. Syrup of hydriodic acid

(2 drachms [7.4 cc.]) is given every four hours as soon as the patient is able to swallow. It is a good expectorant. The patient is placed in a sitting position as soon as possible and is allowed floor privileges and the regular diet the first day after operation.

ABSTRACTER'S COMMENT: The laryngologist is often consulted in cases of vocal injury after thyroid surgery. It may be possible to do decompression or nerve reunion, just as in aural surgery.

I. W. VOORHEES, New York.

OTOSINAL FOCI IN PURULENT MENINGITIS IN CHILDREN. ENRIQUE DELUCCHI, Rev. otorrinolaring. **12**:54 (Sept.) 1952.

The author discusses purulent meningitis in children and stresses the importance of determining the latent focus of infection in either the ear or the sinuses. Most careful examinations should be made and supplemented by x-rays. In doubtful cases surgical exploration is advised. Adequate and early, intensive antibiotic treatment should be instituted. However, it must be remembered that many of these cases are due to a pneumococcus organism which may make the symptomatology rather confusing. DeLucchi reports two groups of patients. In the group, of 21 children, who died of purulent meningitis, 10 of them revealed frank foci either in the ear or in the sinuses. In the group of 15 children who recovered from an attack of purulent meningitis, 8 had a positive focus and 4 others a doubtful focus in either the ear or the sinuses. In conclusion, he stresses the importance of early surgical intervention to avoid not only death but also many serious sequelae, such as cysts, blindness, deafness, hydrocephalus, and idiocy.

PERSKY, Philadelphia.

A NEW SURGICAL PROCEDURE IN BENIGN TUMORS OF THE PAROTID GLAND. DANKO REISSNER, Acta otolaryng. **42**:423 (Aug.-Oct.) 1952.

The author proposes a method of his own for radical procedures on the parotid gland. That which seems basically new in his work is the following: (1) A special open W-shaped circumauricular skin incision is made; (2) the upper branch of the facial nerve is then found where it crosses the malar bone; (3) the remainder of the facial nerve is found and isolated by exposing the external auditory canal and tracing back; (4) the anastomosis between the facial and the auriculotemporal nerve is routinely cut, thus stopping the secretory function of the gland and preventing postoperative fistulas.

ROBERT LEWY, Chicago.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. D. E. S. Wishart, 170 St. George St., Toronto.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Minaki Lodge, Minaki, Ont. Time: June 14-18, 1953.

Fifth International Congress of Oto-Rhino-Laryngo-Broncho-Esophagology

Place: Amsterdam. Netherlands. Time: June 8-13, 1953.
President: Prof. Dr. Eelco Huizinga.
General Secretary: W. H. Struben, Viottastraat 1, Amsterdam-Z
Scientific Secretaries: Dr. J. Bijtel and Dr. P. G. Gerlings.

International Bronchoesophagological Society

President: Dr. Fernand Eeman, Ghent, Belgium.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3.
Meeting: Second International Congress of Bronchoesophagology.
Place: Albert Plage, Le Zoute, Belgium. Time: June 15-16, 1953.

International Congress of Audiology

Place: Leyden, Netherlands. Time: June 5-6, 1953.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

International Course in Paedo-Audiology

Place: Groningen, Netherlands. Time: June 1-4, 1953.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Holland.

Pan American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

Meeting: Fourth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: México, D. F., Mexico. Time: Feb. 28-March 4, 1954.
President: Dr. Justo Alonso.
Exec. Secretary: Dr. Chevalier Jackson, 1901 Walnut St., Philadelphia 3.
President of Congress: Dr. Ricardo Tapia Acuna.

Third Latin-American Congress of Otorhinolaryngology and Bronchoesophagology

Place: Caracas, Venezuela. Time: Feb. 21-25, 1954.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Dean Lierle, Iowa City.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Place: San Francisco.

American Academy of Ophthalmology and Otolaryngology

President: Dr. James Milton Robb, 315 Lakeland Ave., Grosse Pointe 30, Mich.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago. Time: Oct. 11-17, 1952.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Board of Otolaryngology

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Statler Hotel, Boston. Time: May 10-15, 1954.

American Broncho-Esophagological Association

President: Dr. Edwin N. Brayles, 1100 N. Charles St., Baltimore 1.
Secretary: Dr. F. Johnson Putney, 255 S. 17th St., Philadelphia 3.
Place: Statler Hotel, Boston. Time: May 25-26, 1954 (afternoons only).

American Laryngological Association

President: Gordon F. Harkness, 215 Main St., Davenport, Iowa
Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3.
Place: Statler Hotel, Boston. Time: May 20-21, 1954.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Francis E. LeJeune, 49 Audubon Blvd., New Orleans 18.
President-Elect: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Statler Hotel, Boston. Time: May 18-20, 1954 (mornings only).

SECTIONS:

Eastern.—Chairman Dr. Irl H. Blaisdell, 1100 E. Genesee St., Syracuse 10, N. Y.
Place: Hotel Syracuse, Syracuse, N. Y. Time: Jan. 7, 1953.
Southern.—Chairman: Dr. W. W. Wilkerson, Jr., Curtiswood Lane, Nashville 4, Tenn.
Place: Andrew Jackson Hotel, Nashville, Tenn. Time: Jan. 12, 1953.
Middle.—Chairman: Dr. Henry L. Williams, 818 4th St., S. W., Rochester, Minn.
Place: The Drake, Chicago. Time: Jan. 19, 1953.
Western.—Chairman: Dr. Alden H. Miller, 4000 Woking Way, Los Angeles 27.
Place: The Elks Club, Los Angeles. Time: Jan. 24, 1953.

American Otological Society, Inc.

President: Dr. Frederick T. Hill, Professional Building, Waterville, Maine
Secretary-Treasurer: Dr. John R. Lindsay, 950 E. 59th St., Chicago.
Place: Statler Hotel, Boston. Time: May 16-17, 1954.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Harry Neivert, 555 Park Ave., New York 21.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
President-Elect: Dr. Albert D. Reudemann, 1633 David Whitney Building, Detroit 26.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2.
Place: Palmer House, Chicago. Time: Oct. 16, 1953.

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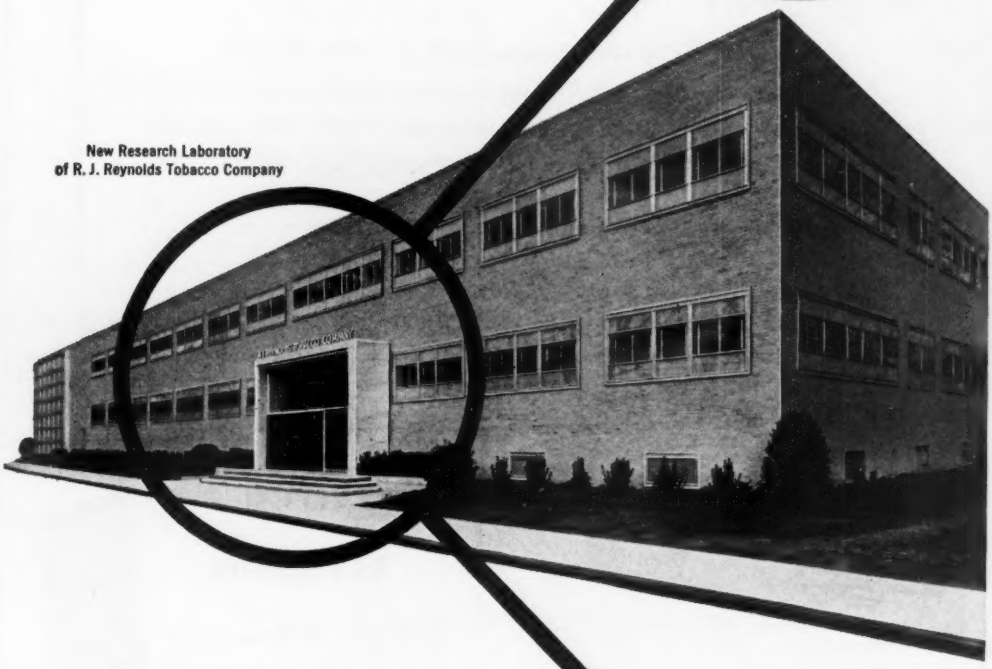

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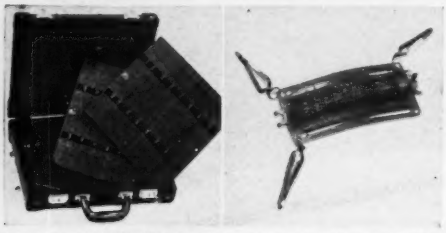

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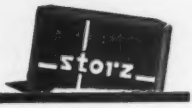

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Today, KENTs are sold in most major U. S. cities. If your city is not yet among them, simply write to P. Lorillard Co., 119 West 40th Street, New York, N.Y., and special arrangements will be made to assure you of a regular supply.

References Cited

a. *J.A.M.A.*, Vol. 103, 1934, p. 318
b. *J.A.M.A.*, Vol. 135, 1947, p. 417

*PATENT APPLIED FOR

Ἔργα τάδε ἰατρ[οῦ Παι]ώνια πρᾶτον ε[---]
καὶ νόον ἰῆσθαι καὶ οἱ πρόπαρ ἢ τῳ ἀ[ρήγην]
μηδ' ἐσιδῆν θυγέην τε παρὲξ καὶ θεσμὰ καὶ ὅρκ[ον]

The reconstructed front of the Sarapion Monument which stood in the Sanctuary of Asesculapius on the south slope of the Acropolis about 220 A.D. Photograph of monument and translation of inscription courtesy of American School of Classical Studies in Athens.

Tradition of service

"These are the duties of a physician: First . . . and to heal his mind and to give help to himself before giving it to anyone (else), and not to look upon (his patient) or make approaches in a manner contrary to divine laws and to the oath. Let him cure not only with (professional) skill but also with blameless character. And as one unfit, though coming in the guise of a helper when he handles lovely maidens and matrons, let him not burn in his breast with desire (in a manner unworthy of a true) physician. Therefore I declare to the godly minded and the pure . . . Possessed of such a mind, like a savior god, let him make himself the equal of slaves and of paupers, of the rich and of rulers of men, and to all let him minister like a brother; for we are all children of the same blood. Therefore let him not hate any one nor hide envy in his heart, nor be lifted up with pride."

Behind the physician stand centuries of the tradition of service. When disaster strikes, in peace as in war, wherever there is sickness or pain, the doctor brings relief and help. ***SONOTONE, an organization dedicated to the service of the hard of hearing, salutes humanity's greatest public servant, the doctor.***

SONOTONE, too, has its own tradition of service. SONOTONE hearing aids are fitted from over 300 possible combinations to match many patterns of deafness as revealed by the audiometric chart. An annual guarantee plan gives the user a unique sense of security. More than 400 permanent offices provide uninterrupted service for SONOTONE wearers.

SONOTONE Models No. 900, 910, 920, 925, 940 and 966 are on the list of AMA Council accepted devices.

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Here's a *taste treat* so different that it has made OLD GOLD the fastest growing of all leading "regular" cigarettes . . . *last year* and *every year* for the past *five years* . . . now yours for extra mildness, extra coolness in KING SIZE.

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Smoke OLD GOLD for a *treat* instead of a treatment, now Regular and KING SIZE!

NEO-SYNEPHRINE®

HYDROCHLORIDE

. . . *reduces nasal engorgement* . . .

. . . *promotes aeration* . . . *encourages drainage*

Supplied in 0.25% solution (plain), bottles of 1 oz., 4 oz. and 16 oz.; 0.25% solution (aromatic), bottles of 1 oz. and 16 oz.; 0.5% solution, bottles of 1 oz.; 1% solution, bottles of 1 oz., 4 oz. and 16 oz.; 0.125 (⅛)% solution, bottles of ½ oz.; 0.5% water soluble jelly, in ⅝ oz. tubes.

1. Van Alyea, O. E., and Donnelly, Allen: Arch. Otolaryng., 49:234, Feb., 1949.

A few drops of Neo-Synephrine 0.25% in each nostril will promptly check mucosal engorgement and hypersecretion, promoting greater breathing comfort over a period of several hours.

The resultant relief to the hay fever sufferer is decidedly gratifying. Prolonged action of Neo-Synephrine makes fewer applications necessary, consequently longer periods of rest and sleep are possible.

Neo-Synephrine does not lose its effectiveness on repeated application and may, therefore, be relied upon to give relief throughout the hay fever season.

Neo-Synephrine is practically free from sting and compensatory congestion; does not appreciably inhibit ciliary activity. Neo-Synephrine has been found relatively free from systemic side effects such as nervous excitation, cardiac reaction or insomnia even when tested on hypertensive, cardiac and hyperthyroid patients.[1]

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